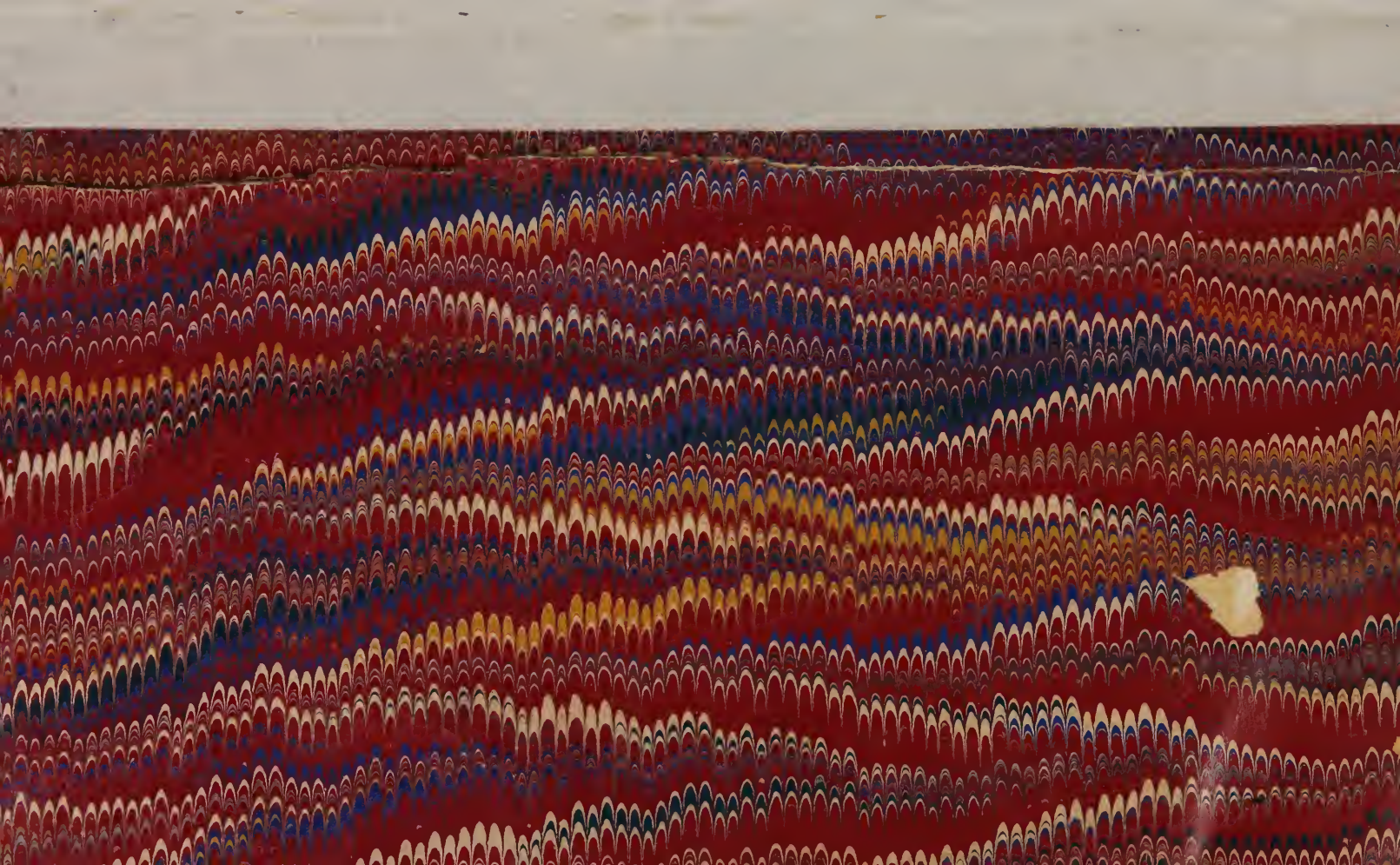

AN

EXPERIMENTAL AND CLINICAL STUDY OF AIR-EMBOLISM.

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AN EXPERIMENTAL AND CLINICAL STUDY OF AIR-EMBOLISM.

SUDDEN and unexpected death during an operation is a calamity which never fails to strike terror to the heart of the boldest surgeon. Although death is a frequent and familiar visitor wherever human beings exist, nevertheless its sudden and unforeseen advent conveys with it more than the usual halo of sadness, and when such a scene transpires in the operating room it leaves impressions which neither time nor space can erase. Disasters of this kind come without warning and usually at a time when least expected. The surgeon who has the misfortune to meet with such an accident is not only destined to burden his memory with the unpleasant remembrances of the incident for the remainder of his days, but in addition he is often made an unjust object of reproach by those who are unable to appreciate the nature of the case. His conscience may be relieved by a favorable verdict regarding his conduct and management of the case before the only competent tribunal, composed of his colleagues and medical press, but that most uncertain of all things, public opinion, will, in all probability, be arrayed against him. In one sad moment the object of his ambition, the ultimate aim of his lifework has suffered irreparable loss. The surgeon who seeks to maintain and advance the interests of his profession as well as his own reputation should familiarize himself with all the causes and conditions which may precipitate such an unhappy result, with a view to adopt and apply timely prophylactic measures. Believing that it is good practice to prepare for war in time of peace, I intend on this occasion to call your attention to one of the most dreaded and, I may add,

one of the most uncontrollable causes of sudden death; I allude to air-embolism.

After consideration of the subject from an historical, experimental, and clinical standpoint, I shall endeavor to point out the conditions remote and direct which give rise to this accident. The different explanations of the immediate cause of death will be discussed and, finally, I shall offer some practical suggestions relating to the prophylactic and therapeutic measures.

By air-embolism, I understand the presence of free atmospheric air within the vascular system during life and in sufficient quantity to give rise to symptoms of obstruction. It is a true embolism inasmuch as the location of the volume of air which constitutes the embolus is always some distance from its point of entrance. The presence of air in a vessel offers the same mechanical obstruction to the flow of blood as a solid substance, and gives rise to the same disturbance of circulation in the tissues supplied by the vessel. An air-embolus differs from an ordinary embolus that, when once introduced into the circulation, it is capable of being broken up or divided by the blood current and the action of the heart, and on this account usually becomes the source of multiple emboli. Pathologically it also differs from a solid embolus, inasmuch as it is more likely to be removed by absorption and is less liable to be followed by thrombosis. Air-embolism is always due to the introduction of atmospheric air into a wounded or injured vein, and, in contra-distinction to the ordinary form of embolism, it is primarily most always formed in the right side of the heart and in the venous system. With a view to study the immediate effects of the presence of a considerable amount of air on the heart and the vessels, it becomes necessary to allude to the experiments which have been made where embolism was artificially produced by the introduction of solid substances into the circulation.

I. *On the Immediate Cause of Death by rapidly Fatal Embolism.*

The most interesting experiments on embolism were made by Virchow, in 1847, and by Panum, in 1854–1855, and although

the conclusions of these experimenters are somewhat at variance, our present knowledge on this subject is based upon the conjoined labors of these distinguished writers.

Virchow was convinced that his experiments were conclusive in showing that complete embolism of the pulmonary artery would invariably prove fatal in a short time, while partial obliteration of this vessel produces either no symptoms at all or only temporary dyspnœa, restlessness, and a sense of oppression. He gives the following explanation to account for the immediate cause of death in cases of complete obstruction in the pulmonary artery by a large embolus.[1] "The first effect of the pulmonary ischæmia is the interruption of the supply of oxygenated blood to the coronary arteries of the heart and the arteries of the body, as well as the stasis of the venous blood in the right side of the heart, the coronary veins, and the veins throughout the body. These conditions result in the arrest of the heart's action in the diastole, the tetanic contractions of the voluntary muscles, the retardation of respiration, the dilatation of the pupils, the protrusion of the eyeballs, etc., and very soon complete death."

Panum, by a series of very ingenious experiments, disproved the assertions of Virchow, that a lack of arterial blood in the coronary arteries produces instantaneous arrest of the heart's action.[2] In a rabbit, where the ventricular contractions had ceased for fully fifteen minutes and where only the right auricle continued to pulsate, he injected a warm black mass, composed of tallow, wax, and soot, into the aorta for the purpose of studying more accurately the anatomical relations of the coronary arteries. The injection penetrated the smallest vessels. The right auricle continued its rhythmical movements for three and a half hours after the injection was made, the heart and lungs having been removed from the body. In a second experiment he divided both pneumogastric nerves in a dog, and then opened the chest and the pericardium and passed a double ligature underneath the innominate artery; after tying the upper ligature

[1] Gesammelte Abhandlungen, p. 297.

[2] Experimentelle Beiträge zur Lehre von der Embolie. Virchow's Archiv, xxv. p. 308.

and drawing the lower tight, the artery was opened between them and a silver tube introduced and secured in the vessel with the proximal ligature. The silver tube was connected with a glass tube by interposing a piece of rubber tubing. To the distal end of the glass tube another piece of rubber was attached and all tubes filled with oil, which was kept from leaving the tubes by a clamp. The aorta was compressed with a spring forceps above the origin of the innominate artery and the same black mass was injected through the tubes into the aorta. The oil and black mass entered the vessel, and after closing the aortic valves filled the coronary arteries, which were found completely blocked with the foreign substance. The movements of the heart were carefully observed before, during, and after the injection was made. Before the injection, the contractions were regular, 80–90 per minute; during the injection, on account of the higher temperature of the injection (45° C.), the contractions became more rapid, and in the left side of the heart, which was distended with blood, they were less forcible, an occurrence which could be readily accounted for by the mechanical obstruction to the outflow from the left ventricle. All the chambers of the heart continued to contract for five minutes after the injection was complete. Six minutes after the injection was made, the contractions of the left auricle ceased. The movements of the ventricles were feeble, but could be plainly seen. The rhythm of the ventricular contractions grew slower than the contractions of the right auricle, and at the same time were less regular. After twenty-five minutes the right auricle pulsated 48, the ventricles 24 times per minute. Five minutes later the pulsations of the auricle and ventricles were the same in frequency. Forty minutes after the injection the ventricles contracted 24 times per minute to 8 auricular pulsations. Fifty minutes after the injection the ventricles made 23 rhythmical movements to 3 of the auricles. After one hour the movements of the heart again became regular, inasmuch as the auricles and ventricles pulsated 13 times per minute, and in such a manner that the movements of the ventricles followed immediately after the contraction of the auricles and were followed by a long dias-

tolic pause. Seventy-five minutes after the injection the movements of the left ventricle ceased, while the right half of the heart contracted regularly 8 times per minute. Two minutes later the ventricle contracted only twice to 8 movements of the auricle. Ninety minutes after the injection, the right ventricle ceased to beat, while the right auricle continued to contract for 6 hours and 10 minutes after the injection, making toward the last only 1 movement per minute. After all pulsations had ceased for a while they were renewed by blowing upon the heart. These contractions continued for seven and a half hours after the injection, and even after they had ceased for a second time they were again excited by mechanical irritation. During this observation the heart was kept at a temperature of 12.5 to 13° C. under a glass bell in which the air was saturated with moisture. An examination of the heart showed that the coronary arteries were completely distended with the black mass, and that the capillary vessels and coronary veins were blocked with oil. A few drops of oil, but nothing of the black mass were found in the right auricle. The aorta near the heart was filled with the injection material, and the aortic valves were so completely closed that nothing had penetrated into the ventricle. In two other instances the coronary arteries were made impermeable in a similar manner, and the contractions of the heart were temporarily arrested by electric irritation of the pneumogastric nerves. This experiment was repeated in both animals more than 20 times, and always with the same uniform result. If the electric stimulation was continued after the heart had ceased to contract, the movements were again excited, but this always required a continuation of the current for at least a minute. If the electrodes were removed after the heart ceased to act, it required twenty seconds before the contractions were re-established, the movements being always more rapid than before the irritation was applied. From these experiments we are forced to conclude that embolism of the coronary arteries is insufficient to produce instantaneous arrest of the heart's action. Virchow quotes Erichsen as having observed prompt cessation of the movements of the heart after ligation of the coronary arteries, but Panum doubts the possi-

bility of performing this operation upon the heart of a living animal.

The same observer studied embolism of the pulmonary artery by injecting an emulsion of gum-arabic, in which were suspended small pellets of black wax, into the jugular vein of a medium-sized dog. Eight c. ctm. of the emulsion were injected. All signs of life and all reflex movements ceased three minutes after the injection. After death the large vessels were tied, in order to ascertain the exact quantity of blood contained in each side of the heart. The right side contained 112.35 grms. of dark-colored blood, the left contained only 6.45 grms. In another experiment he injected coarsely powdered charcoal in suspension into the jugular vein of a dog. The time which elapsed between the injection and cessation of life was longer than in the preceding case, consequently the left heart contained a larger amount of blood, although the quantity was small when compared with that of the right side of the heart. In all cases of death resulting from embolism of the pulmonary artery, the amount of blood found in the left side of the heart is proportionate to the completeness of the obstruction in the pulmonary artery. The left side of the heart is never found completely empty, as the labored respiratory movements will force the blood, which is present in the pulmonary vein and its branches, into the left side of the heart. If instead of using small emboli large plugs are injected, as was done by Virchow, the blood contained on the distal side of the obstruction will pass through the pulmonary circulation and reach the left side of the heart, consequently in such cases more blood will be found in the left ventricle. Panum asserts also that the cessation of the heart's action does not invariably take place so early that it can be considered as the primary and direct cause of death. As a rule, he found the heart pulsating after the death struggle had been initiated from arrest of innervation from the cerebro-spinal centre. In some instances the heart continued to pulsate after all signs of animal life emanating from the brain and spinal cord had ceased. Shortly after respiration was arrested the heart did cease to pulsate, and, as Virchow has stated, in the diastole.

According to Panum, the cessation or continuation of the heart's action exerts no influence for good or evil in cases of extensive embolism. He claims that if the cessation of the heart's action takes place as one of the first effects after embolism of the pulmonary artery, as was noted in Virchow's first case, it must be regarded, under certain circumstances, as being the result of irritation of the pneumogastric nerves, so much more so, as the heart in the case referred to again began to pulsate after the thorax was opened. As a rule, the heart's action is arrested by distension of the right ventricle.

Other observations tend to show that the distension of the right ventricle is the cause. The excess of carbonic acid gas and the diminished supply of oxygen must also be taken into account. Other experiments have demonstrated that carbonic acid, in concentrated form, injected into the heart after its removal from the chest readily leads to diastolic paralysis, and that the organ commences to beat again when exposed to air. The arrest of the heart's action is due to mechanical dilatation and the presence of an excess of carbonic acid. The first and most constant symptom resulting from sudden and extensive embolism is a high degree of anæmia in all visible parts of the body. On post-mortem examination the white substance of the brain is completely bloodless, especially if small and numerous emboli have been injected. This general anæmia is followed by tetanic stretching of all extremities, involuntary discharges, and deep, convulsive, inspiratory movements. Ligature of both carotid arteries does not produce such an intense ischæmia of the brain. If the vertebral arteries are ligated at the same time, the tightening of the ligature of the second vertebral artery produces syncope and convulsions, but the symptoms are less intense than after sudden, fatal embolism of the pulmonary artery. Panum also induced cerebral anæmia by injecting black pellets of wax, suspended in an emulsion, into the crural artery of a dog, throwing the injection in a central direction through a catheter which had been passed into the artery near the heart, producing thus multiple embolism in all of the smaller arteries. The animal lost only a few drops of blood, and no air entered.

The animal was taken immediately with tetanic convulsions, involuntary discharges, and all organs accessible to the eye presented an extremely anæmic appearance. All reflex symptoms were arrested after one to two minutes. Two other experiments were followed by the same results. In all cases the small wax pellets were found in large number in the small vessels of the brain, as well as in all other parts of the body. In four other dogs cerebral embolism was avoided by introducing the catheter only as high as the ribs, and by injecting slowly. During the injection a peculiar tremor was observed, which affected the muscles of the lower extremities; this, however, soon ceased, and gave way to complete paralysis of both motion and sensation, as well as complete arrest of all reflex movements. One of the animals survived the experiment 22 hours, the second $9\frac{1}{2}$ hours, the third 6 hours, and the fourth 5 hours. The small vessels of the spinal cord were found obstructed by the small wax pellets, the vessels between the emboli and the heart were much dilated, and showed many small extravasations. The spinal cord was the seat of red softening, which was more conspicuous the longer the life of the animal was prolonged. The spinal cord above the middle of the dorsal region and the brain were normal in appearance, although scattering pellets were found here also.

It will be seen that Panum, in contra-distinction to Virchow, attributes the immediate cause of death in cases of rapidly fatal embolism to acute cerebral anæmia.

From a study of the literature on air-embolism, it is evident that the immediate cause of death has been assigned by different pathologists to one of the following conditions:—

1. Mechanical dilatation of the heart and paralysis of the organ in the diastole.
2. Acute cerebral ischæmia.
3. Asphyxia, resulting from mechanical obstruction to the passage of the blood through the pulmonary circulation.

As we shall see further on, death from air-embolism is not

always produced in the same manner; the mode of dying varies and is modified:—

1. By the amount of air admitted.
2. The time which has elapsed between the ingress of air and the fatal issue.
3. The location and distribution of the emboli.

II. *History of Air-Embolism.*

Surgeons and pathologists have for a long time been aware of the deleterious effects of free atmospheric air in the vascular system. The danger attending the forcible insufflation of air into the veins of animals was well known to many of the earlier physiologists. Among the first to study the effects of the introduction of air into veins may be mentioned Redi, Wepfer, Camerarius, de Heyde, Harder, Bohnius, Boerhaave, Lancisi, Morgagni, Valsalva, Bichât, and Nysten. As early as 1667 Redi killed animals by intravenous injections of air. He observed during his experiments that the pulse became intermittent, an occurrence which he attributed to the passage of a large air bubble through the heart. His followers who repeated the experiments soon discovered that after forcible insufflation of air into veins the air became diffused, inasmuch as at the post-mortem examinations they found it present in the right auricle, the coronary vessels, and in the shape of air bubbles, in the smaller vessels.

Merg made the observation that in opening the abdomen of a dog and puncturing the vena cava above the origin of the emulgents, as the vein became emptied of blood it filled with air which ascended with the blood current and entered the right side of the heart. Haller witnessed the same phenomenon in cold-blooded animals after wounding some of the large venous trunks. He has shown that it was from this source that the air was derived which Redi, Caldesi, and Morgagni had seen circulating in the vessels of the same animals. He claimed that air is never seen in vessels when the necessary precautions are exercised to prevent its introduction through a wounded vein.

Nysten found, by injecting air slowly into a vein, so as not to produce the death of the animal, that the coloring of the arterial blood was rendered imperfect. He satisfied himself that this change was not owing to the embarrassment of respiration. Insufflation of oxygen had no effect in preventing or correcting this change of color in the arterial blood. The literature on insufflation of air into veins is quite prolific and this subject cannot be justly passed over without an allusion to the following names:—

Blochmann. Aër in venis causa mortis. Dresden, 1843.
Bouillaud. De l'introduction de l'air dans les veines. Paris, 1838.
Gain. De aëris ingressione in venas. Berlin, 1865.
Maguin. Étude expérimentale sur l'introduction forcée et sur l'entrée spontanée de l'air dans les veines. Nancy, 1879.
Méric. Recherches sur l'introduction de l'air et des gaz qui le constituent dans le système veineux. Paris, 1866.
Valkenhoff. De aëris in venas ingressu ejusque effectu lethali. 1840.
Laborde. Effets de l'introduction de l'air dans la circulation artérielle. Compt. Rend. Soc. de Biolog. Paris, 1873.

These names are intimately associated with the experimental part of the history of air-embolism.

It was not long after the deleterious effects of free atmospheric air in the veins of animals had been studied experimentally before the same symptoms were observed in man by the accidental admission of air into wounded veins during operations, and in some of the first cases the presence of air in the veins and right side of the heart was demonstrated by post-mortem examination. Although a number of honest and reliable surgeons, prominent among them Velpeau and Fergusson,[1] have denied that a sufficient amount of air can be admitted through a wounded vein to produce sudden death, this assertion is no longer tenable in the face of such a large number of well-authenticated cases as have been recorded in surgical literature by equally conscientious and competent observers. Since the publication of the first well-authenticated case observed by Beauchène and described by

[1] Lettre sur l'introduction de l'air dans les veines de l'homme. Gaz. Méd, pp. 113–121. Paris, 1838.

Magendie the following authorities, placed in alphabetical order, have reported similar cases.

Assmus. Zur Casuistik des Lufteindringens in grössere Venenstämme. Med. Zeitung, xi. p. 104. Berlin, 1842.

Amussat. Introduction de l'air dans les veines. Bulletin Acad. de Méd., Paris, 1836, i. pp. 894, 899; 1837–38, ii. pp. 363, 461.

Barlow. An Attempt to Remove a Tumor on the Neck; Entrance of Air in Vein; Sudden Death. Med. Chir. Trans., xvi. pp. 28–35, 1830.

Chassaniol. Observation de l'entrée de l'air dans les veines pendant l'amputation du bras, dans son articulation scapulo-humérale. Union Méd., viii. p. 428. Paris, 1869.

Clémot. Lanc. Franc., tom. i. p. 357, 1830.

Coolidge. Case of Sudden Death from Entrance of Air into the Jugular Vein. New York Med. Gazette, i. p. 305, 1841–2. Also New York Med. Journal, vol. ix. pp. 199–201, 1847.

B. Cooper. Case of Alarming Syncope from the Admission of Air into a Vein, during Amputation of the Shoulder-Joint. London Lancet, i. pp. 448–451, 1843.

Cormack. Case of Death from the Entrance of Air by a Rigid Vein in the Neck, opened accidentally by a Seton-Needle. London Med. Journal, 1850.

Delaporte. Extirpation l'une tumeur située au cou; introduction de l'air dans le système vasculair. Bulletin Acad. de Méd., i. p. 132. Paris, 1836.

Delpech. Mém. des hôpitaux du midi, No. 16, p. 231, 1830.

Fischer, H. Ueber die Gefahren des Lufteintritts in die Venen während einer Operation. Volkmann's Sammlung klin. Vortraege. Chirurgie, No. 34.

Gunn. Syncope from Entrance of Air into the Facial Vein. New York Medical Journal, p. 356, 1852.

Heckford. Four Cases of Entry of Air into the Circulation. Medical Times and Gazette, i. p. 137. London, 1867.

Koestlin. Ein Fall von Luft im Herzen. Med. Correspondenzblatt d. württ. ærztl. Ver., xxviii. pp. 316–321. Stuttgart, 1857.

De Lavacherie. De l'opportunité d l'extraction des tumeurs du cou non susceptibles de resolution; reflexions sur l'introduction de l'air dans le cœur par des veines ouvertes accidentellement. Mém. Acad. Roy. de Méd. de Belge, ii. pp. 305–376. Bruxelles, 1849.

McPharlin. Death from Entrance of Air into the Veins in a Case of Compound Fracture. Hosp. Gazette, iii. p. 20. New York, 1878.

Massart. Etude nouvelle sur l'entrée de l'air dans les veines, dans les cas de plaie ou d'opération chirurgiçale. Annales Soc. de Méd. d'Anvers, xv. pp. 5, 57, 113, 1854.

Mercier. Journal des connaiss, p. 108, September, 1836.

Meyer, F. Case of Injury of the Vena Jugularis Interna; Entrance of Air; Sudden Collapse; Recovery. Med. Archives, iii. pp. 408–410. St. Louis, 1869.

Miner. Tumor in the Neck; Admission of Air into the Vein; Death. Buffalo Med. and Surg. Journal, pp. 336–338, 1864.

Mirault. Thèse. Paris, 1832.
Mott. Entrance of Air into the Facial Vein. Medico-Chir. Trans., 1830.
Piachaud. Mort par introduction de l'air dans une veine pendant l'ablation d'une tumeur du sein avec ganglions dans l'aisselle. Echo Méd., p. 768. Neuchât, 1857.
Porter. On the Entrance of Air into the Veins as a Cause of Death. Journal American Med. Asso , iii. No. 20.
Rauch. Lufteintritt in einen verletzten grösseren Halsvenenast und seine Folgen. Oest. Med. Wochenschr., pp. 199–201. Wien, 1845.
Roux. Journ. Hebdom., ii. p. 64, 1833.
Schmid. Das Eindringen von Luft in eine Vene während einer Operation am Halse. Corresp. blatt d. württ. ärzt. Ver., xxi. p. 53. Stuttgart, 1851.
Schweickhart. Eindringen von Luft in die Venen; Tod durch Gehirnschlag. Mitth. des badischen ärzt. Ver., vi. pp. 69–71. Karlsruhe, 1852.
Smith, R. W. Abscess behind the Pharynx; Entrance of Air into Veins. Dublin Quarterly Journal Med. Sciences, xxv. p 497, 1844.
Tadlock. Entrance of Air into Divided Internal Jugular Vein; Ligation; Recovery. American Journal of Medical Sciences, p. 280, 1875.
Ulrich. Tod durch Eintritt von Luft in die Venen. Med. Zeitschrift des Vereins für Heilkunde, p. 132, November, 1834.
Warren, J. C. Two Cases of Accidents from Admission of Air into the Veins during Surgical Operations. American Journal of Medical Sciences, pp. 545–548, 1832.
Warren, J. M. Tumor connected with the Sartorius Muscle; Secondary Cancer of Breast; Operation; Entrance of Air into the Vein; Recovery. Surgical Observations, p. 529. Boston, 1867.
Wattmann. Prager Viertelj., ii. p. 191, 1844.

This list is, of course, not complete, nor does it represent all cases of accidental introduction of air during operations; but the names which are quoted ought to be accepted as sufficient guarantee by the most skeptical that the fear of this accident is not a myth but a reality substantiated by many a sad experience.

III. *Intravenous Production of Air.*

Spontaneous production of air within the bloodvessels of recently deceased persons has been repeatedly observed, and to it has been assigned one of the causes of sudden death. That the air thus produced is a direct product from the blood appears to be negatived by the fact that its occurrence has usually been traced in connection with sudden and exhaustive hemorrhages. It is, in fact, in persons who have died from hemorrhage, that

air has been found in greatest abundance in the veins. Lieutand[1] reports the case of a girl who died suddenly in a state of syncope, after having been repeatedly bled, and in whom the cerebral veins and choroid plexus were found impacted with air. M. Rerolle[2] has published several cases of the kind, where profuse hemorrhage had existed; in one of fatal epistaxis, the heart, arteries, and veins contained large quantities of air. Dr. Graves has noticed emphysema of the abdominal parietes in a sufferer from repeated attacks of epistaxis. M. Rerolle conjectures that, in such cases, air is absorbed by the radicles of the pulmonary veins—the air would have no claim to be considered adventitious.[3] It is, however, more logical to assume that, inasmuch as in almost all cases the supposed intravenous origin of air took place consequent upon severe losses of blood, hence likewise in connection with loss of continuity of the vascular system, that, owing to the sudden loss of intravascular pressure, the air may have been aspirated through the openings of some of the bleeding vessels. The quantity of air found in these instances has been so small that it has been impossible to make a chemical examination to determine its identity with atmospheric air. In cases where air was found in blood without loss of continuity of the vessels, it is not impossible that the supposed air was not atmospheric air, but a gaseous product liberated from the blood or generated in the tissues, producing a gas-embolism which interferes with the function of circulation in a similar manner as when the obstruction is caused by atmospheric air.

IV. *Effect of the Heart and Respiration on the Venous Circulation.*

As the state of the intravenous blood pressure constitutes the most important element both in the prevention and causation of aspiration of air into veins, this subject must be briefly alluded to in order to determine the conditions which act as exciting causes. For the most reliable and comprehensive information

1 Hist. Anat., Med. Obs. 55. 2 Thèse de Paris, No. 129, 1832.
3 Todd's Cyclopædia of An. and Physiol., vol. iv. part i. p. 145.

on this subject we are indebted to Jacobson.[1] The observations were made on sheep. To determine the effect of the heart's action upon the venous circulation he measured the blood-pressure in veins with the manometre. These measurements were made on veins in close proximity to the heart, as the lower portion of the jugular and subclavian, as all attempts to approach nearer the heart seriously impaired the normal physiological conditions of the respiratory and circulatory organs. This measurement gave the following results :—

In the left vena anonyma . . .	— 0.1 mm. Hg.
" " right " jugularis . . .	+ 0.2 " "
" " right " subclavia . . .	— 0.1 " "
" " left " jugularis . . .	— 0.1 " "
" " left " subclavia . . .	— 0.6 " "

The following are his observations on some of the more distal veins in the same animal :—

In the external facial	+ 3.0 mm. Hg.
" " internal "	+ 5.2 " "
" " brachial	+ 4.1 " "
" a branch of the same	+ 9.0 " "
" the crural vein	+ 11.4 " "

The experiments of Ludwig and Mogk, although made in a similar manner, led to more variable and inconstant results; at one time they found the blood-pressure in the crural vein 6.8 mm. Hg., while on another occasion under similar circumstances it measured in the same vein only 1.9 mm. Hg. Donders explains this want of uniformity to the respiratory movements of the chest, believing that the aspiratory movements of the chest affect the venous circulation more than the *vis a tergo* from the capillary system. Poisenille claimed that in his experiments the manometre was affected by the respiratory movements of the chest only when it was inserted into veins in close proximity to the heart, as in the lower portion of the jugular and the external iliac veins, while in more distant veins the column of

[1] Dr. Heinrich Jacobson. Ueber Blutbewegung in den Venen. Virchow's Archiv, xxxvi. p. 80.

mercury was not affected by the movements of the chest. Volkmann obtained the following measurements:—

In the facial vein of a goat	41	mm. Hg.
" " jugular vein of a goat . . .	18	" "
" " metatarsal vein of a calf . . .	27	" "
" " jugular vein of a calf . . .	21.5	" "
" a subcutaneous vein of the neck of a horse	44	" "
" the jugular vein of a horse . . .	21.5	" "

Magendie found the blood-pressure in the external jugular vein of a dog 18 mm. Hg., and in the crural vein 50 mm., although the measurements of the intravenous blood-pressure taken by different observers are at great variance, and although their figures are indicative of the opinions held by the different experimenters as to the effect of respiration upon the return of venous blood, yet they all agree in locating the minimum degree of intravenous pressure in the veins nearest the heart. The effect of respiration on the venous circulation was thoroughly investigated by Magendie. He introduced an elastic tube into the internal jugular vein, and observed that blood would escape only during expiration. The same experiment was made on the crural vein by directing the tube towards the heart, followed by the same result. The suction force exerted during inspiration was sufficient to counter-balance the auricular contractions. In making these experiments air was frequently drawn into the heart during forcible inspiration. Barry introduced through the jugular vein of a horse a bent tube of glass, one extremity being passed into the right cavity of the heart, or the vena cava, and the other into a vessel containing a colored fluid. He found that with each act of inspiration the liquid rose in the tube, demonstrating the effect of a notable suction force. He found that this suction force was increased by preventing the entrance of air into the chest by the trachea. He was of the opinion that this force from the chest was exerted not only in the large veins near the heart, but throughout the entire venous system.

Schweinburg[1] has studied the effect of respiration on the

[1] Die Bedeutung der Zwerchfellcontractionen fuer die respirat. Blutschwankungen. Du Bois-Reymond's Archiv, 1881, p. 475.

circulation by producing paralysis of the diaphragm by section of the phrenic nerves. He states that when diaphragmatic respiration has been artificially arrested, the difference of blood pressure observed during respiration ceases entirely or nearly so. From this he concludes that the action of the diaphragm causes to a certain extent these differences. Even after opening the abdominal cavity the difference in blood-pressure is very slight. As the principal cause in abolishing the effect of respiration upon the circulation he looks upon the compression of the abdominal vessels during inspiration, causing the increase of blood pressure during inspiration and its diminution during expiration by diminishing intra-abdominal compression. If the jugular vein in an animal is exposed, direct observations show conclusively that the direct influence of inspiration cannot be felt much beyond these vessels. The flaccidity of the walls of the veins will not permit the extended action of any suction force, but the flow of blood in the distant veins is accelerated by the intermittent emptying of the veins by the respiratory act. Barry and Donders ascribe to the aspiratory function of the chest, the principal motor in the return of the venous blood. Donders estimated the aspiratory force of the inspiratory movements of the chest at 7 mm. Clinical observation and experimental research have established the fact that the venous circulation is directly influenced by respiration within a certain area, and that aspiration of air in the majority of cases takes place in those veins thus affected, thus constituting the justly and much dreaded "danger-zone." Instead of speaking of the effect of respirat on on veins as a cause of aspiration of air, some authors speak of the vein pulse, and limit the danger-zone to such veins which pulsate. Under certain circumstances the pulsations of the arteries are communicated directly to the veins through the capillaries. In such instances it is necessary that the arterioles are relaxed, as has been ascertained by Bernard in observing the circulation in glands during their physiological activity. If a vein be opened in a gland during its physiological activity, the blood retains partly its arterial hue and escapes in intermittent jets, as from a divided artery. According to recent

physiological investigations veins continue to pulsate independently of the arterial system and the cerebro-spinal centres. Luchsinger[1] examined the venous pulsation in the wings of bats. Contrary to Schiff's observations he found it independent of the central nervous system. Division of the brachial flexus and separation of all tissue connections between hand and body, with the exception of the vessels, did not arrest it. If artificial circulation was established in the organ after amputation rhythmic venous contractions would be seen even twenty hours after death. Intravenous pressure was found to be of great importance in these experiments; as soon as it was increased the vein began to pulsate. The seat of these rhythmic contractions Luchsinger placed in the walls of the vessels, or rather in their muscular structures. They are probably regulated by the central nervous system. Slight increase of warmth and electric tetanization accelerate the contractions. High temperature causes diastolic stasis. Nitrite of amyl increases the pulsating only to arrest it later. Schiff has since satisfied himself that these pulsations continue after division of the brachial plexus and ligature of the vessels, and even in the veins in detached pieces of the bat's wing.

Brunton[2] has made the same observations on man in regard to the effect of increased intravascular pressure in producing venous pulsations in the larger veins. He finds that pulsation of the jugular vein is sometimes confined to one side, the left one. In one of his cases the jugular on the left side was much more distended than the right jugular, the distension increasing whenever the vein was compressed just above the clavicle. Whenever this compression was repeated in the rhythm of the pulse, the increase and decrease of the blood in the vein assumed the character of pulsation, and for this reason the author has arrived at the conclusion that the venous pulsation in such instances is caused by compression of the vena anonyma by the aorta. All cases of unilateral jugular pulsation observed by

[1] Von den Venenherzen in der Flughaut der Fledermaäse. Pflueger's Archiv, 1881, vol. xxvi.

[2] On Pulsations in the Jugulars and other Veins. Medical Press and Circular, July 2, 1879.

Brunton occurred in anæmic women. In one of these the pulsation took place only while the patient was affected by some emotional excitement, in another only during expiration. In rabbits the author has repeatedly observed rhythmical contractions of the pulmonary veins, the vena cava inferior, and the portal vein, occurring immediately after the death of the animals. These pulsations were present either after complete cessation of the heart's action, and sometimes even before death, and, as the pulsations were more frequent than the heart's action, it was plain that they occurred independently from any contraction of that organ. In consequence of long-continued pressure on a vein the author has seen tonic contractions to take place, especially in smaller veins, and this may explain the cause of some of the irregularities of the circulation and subsequent transudation.

Riegel[1] has made vein pulsation a special subject of investigation, and as the result of his researches he has come to the following conclusions: 1. There exists in the normal condition a pulsation of the jugular vein. 2. This normal pulsation is always anadicrotic, *i. e.*, its wave rises in two distinct intervals. The anadicrotic wave corresponds, in contra-distinction to the pulsation of the carotids, to the disatole of the heart. The short catacrotic line or wave corresponds to the systole, the anacrotic to the diastole of the heart. Synchronous with the systole, the contents of the vein are emptied into the heart, while during the diastole stasis takes place in the veins.

King,[2] in his interesting essay "On the Safety-valve Function in the Right Ventricle of the Human Heart," demonstrates the existence of venous pulsations in the veins of the hand, the median veins of the forehead, and the external jugular which he observed after a full meal. The pulsations were made plainly visible by taking a delicate thread of sealing wax about two inches in length, one end of which was fixed across the vein with a little tallow so as to make a long and excessively light lever, capable of indicating a very slight movement in the vessel.

[1] Zur Kenntniss von dem Verhalten des Venensystems unter normalen und pathologischen Verhæltnissen. Berl. Klin. Wochenschrift, 1881, No. 18.

[2] Guy's Hospital Reports, 1837, p. 108.

The movements of the lever produced by the vein pulse corresponded in frequency with the pulsations of the arteries in the same vicinity, but did not correspond in time as the venous pulse followed the arterial systole, showing conclusively that it was not due to the impulse of an adjacent artery. The pulsations could only be caused by the arterial wave being continued to the veins through the capillary vessels. In certain pathological conditions independently of valvular lesion of the heart he noted a marked increase in the venous pulsations in the dorsal veins of the hand and other vessels distant from the heart. The subject of the vein pulse affords an interesting topic in physiology, but in connection with this paper it is only mentioned in order to show that the intravenous tension is only slightly affected by it, and consequently it can exert no direct influence in causing aspiration of air into veins. The venous pulsations which directly influence the return of the venous blood to the right side of the heart occur synchronously with the movements of respiration and are observed only in the veins which are in close proximity to the heart, and in venous channels with firm unyielding walls. The introduction of air can only follow in wounds of vessels where the intravascular pressure is subjected to great variations either from normal anatomico-physiological conditions or the result of pathological alterations. All causes which prevent a prompt collapse of the walls of a wounded vein must be considered as predisposing causes, while all conditions which tend to produce a vacuum in the wounded vein act as determining causes. The location of the former corresponds to the point of injury, while the latter are always represented by the aspiratory action of the chest during inspiration.

V. *Aspiration of Air into the Superior Longitudinal Sinus.*

Nearly all of the older physiologists were of the opinion that aspiration of air into veins could only take place in vessels which were in close proximity to the heart and within reach of the venous pulse. Mery claimed that the effect of thoracic

aspiration on the venous circulation extends to the sinuses of the dura mater and the venous channels in the diploë of the cranial bones. Bernard was aware that air might enter the sinuses in case these structures were wounded, as this accident occurred a number of times in his experiments on animals where the superior longitudinal sinus was opened for other purposes. He believed that the air, after entering the sinus reaches the heart through the vertebral veins and the vena azygos. Death in such instances took place in eighteen minutes, while forty-five minutes were required if death resulted from hemorrhage alone.

Volkmann's case, reported in another part of this paper, demonstrates to a certainty that death may be caused by the entrance of air through a wound of the longitudinal sinus, and, although this is the only authenticated case on record, similar cases have undoubtedly occurred before, but the real cause of death was not recognized, and the fatal result was attributed to some other source. This subject of aspiration of air into the longitudinal sinus was made the object of experimental inquiry by Genzmer, one of Volkmann's assistants.[1]

The experiments were made on dogs, as this vessel in rabbits was found too small for the operation. Nine experiments were made. The animals were made partially insensible by morphine injections. The skull was exposed by an incision which was carried from the occipital bone to the forehead; with a small straight chisel, a section of bone about 6 cm. square was mapped out by cutting through the external table, anteriorly to the prominentia occipitalis externa, and which was completely detached with a hollow chisel. The dura mater having thus been freely exposed the posterior portion of the longitudinal sinus, which was about 2 mm. in width, was made accessible about its middle. Between two small hooks the sinus was made tense and divided transversely, carefully guarding against injury to the subarachnoidean space. In some of the experiments the

[1] Exstirpation eines faustgrossen Fungus duræ matris, tödtlich verlaufen durch Lufteintritt in den geöffneten Sinus longitudinalis. Verh. d. deutschen Gesellschaft f. Chirurgie, vol. vi. p. 32.

wound was kept patent by making traction on its margins with the hooks, in others this precaution was unnecessary as the edges of the wound retracted sufficiently to keep it open. For several minutes after incision the bleeding continued profusely; the blood was quite red and escaped with some degree of force, the pulsations being plainly visible and synchronous with the heart's action. The stream was also perceptibly increased and diminished with the respiratory movements of the chest. After a few minutes had elapsed the hemorrhage became less profuse. In case the animal died, the heart and lungs were removed, after carefully tying the large vessels so as to prevent the escape of air from the heart. To secure accuracy in ascertaining the presence of air in the heart, this organ was opened under water when the rising bubbles would indicate its presence. In three cases, in two of them the animal breathed through a tracheal canula, the double rhythm in the blood column was lost soon after the sinus was opened, and the blood continued to flow until the animal died, which was usually the case after thirty-five, forty, and fifty-three minutes, the stream from the peripheral end of the sinus growing constantly less during this time. In all of these cases the central end of the sinus was completely filled with a thrombus, and no air was found in the heart. In two other cases the double rhythm continued until life was extinct, which was the case after twelve and nineteen minutes. After the first two or three minutes the bleeding diminished, and, by removing the blood from time to time with a sponge, it could be seen how air was aspirated during inspiration through the gaping wound. During forcible expiration, or on compressing the chest, air bubbles escaped with the blood from the wound, from the proximal end of the sinus. As the bleeding diminished air aspiration became more copious and more frequent. An examination of the cadavers of these animals revealed that the right side of the heart contained air and spumous blood. In the next two cases artificial dyspnœa was produced, in one instance by dividing both pneumogastric nerves, in the other by closing the tracheal canula through which the animal was breathing. In the first case air entered early and the animal died in sixteen

minutes; in the second case air entered freely during the forcible inspiratory efforts; the animal died in twenty-four minutes. In both of these cases air was found in the right side of the heart and in the subpleural vessels. In the last two experiments the animals were killed fifteen and sixty minutes after the sinus was opened, by puncturing the brain with a needle. In the first case a considerable amount of air was found in the right side of the heart, and in the second case the amount of air contained in the right side of the heart was less, the apparent difference being due to the presence of a thrombus in the central end of the sinus in the last case which prevented further ingress of air.

In recapitulation it may be stated that in six out of nine experiments air entered the longitudinal sinus, thus proving conclusively that wounds of this great reservoir of venous blood are not only dangerous from the loss of blood, thrombosis, and inflammation, but may also become the direct cause of sudden death by admitting air into the venous circulation.

VI. *Experiments.*

These experiments were made by the writer for the purpose of ascertaining more fully the conditions which determine the entrance of air into a wounded longitudinal sinus, and, at the same time, to obtain reliable information concerning the prophylactic measures as well as to determine the best methods of arresting hemorrhage in wounds of this vessel. All operations were made under antiseptic precautions; when not specified, no anæsthetic was used. The field of operation was cleanly shaved, and the surface thoroughly disinfected with a five per cent. solution of carbolic acid; during the operations, frequent use was made of the irrigator, using a warm two per cent. solution of the same antiseptic. When the animal survived the operation, the wound was closed with continued catgut sutures, dressed with iodoform, and a compress of salicylated cotton retained by a roller bandage. The operation consisted in making a longitudinal incision in the median line of the skull, reaching from the external occipital protuberance to near the upper extremity of

the frontal sinuses. The soft parts with the periosteum were separated and reflected on each side so as to lay bare the bone over a sufficiently large area for the ready use of the bone-cutting instruments. A medium-sized trephine was applied over the middle of the longitudinal sinus, and the button of the bone carefully removed so as to prevent injury of the underlying vessel. The enlargement of the circular aperture was effected with a hollow chisel and Luer's bone-forceps. The opening in the bone was made of an oval or oblong shape with the longest diameter parallel to the sinus, in order to bring into view a large extent of the vessel with a minimum destruction of the cranial vault.

Experiment No. 1.—Small Skye terrier, weight 12 pounds. Ether used as an anæsthetic. Longitudinal sinus laid bare to the extent of one and one half inches by an oval opening in the skull. Copious hemorrhage from a vein leading into sinus which was arrested after ligature. Two catgut ligatures were placed underneath the sinus about one-half inch apart, and the vessel cut transversely between them. The bleeding was very copious, the blood escaping in jets synchronous with the heart's impulse, the flow was also distinctly increased and diminished by the respiratory movements of the chest. During inspiration the stream was diminished while expiration was always attended by a decided increase in the force of the jet and the amount of bleeding. No air was seen to enter, although the hemorrhage had been very profuse and continuous for a considerable length of time. As it was intended by this experiment to prove that sudden obliteration of the longitudinal sinus is not incompatible with life, the distal ligature was tied with the effect of nearly but not completely arresting the hemorrhage, as some blood escaped from the proximal end of the vessel. It was now expected that air would be more prone to enter through the gaping wound in the sinus as the blood pressure from the distal end of the vessel had been arrested by the ligature, but as this accident did not take place after a few minutes the second ligature was tied, and the wound in the skin united with the continued catgut suture, and the antiseptic compress applied. The animal showed no other symptoms except great prostration from the sudden and profuse loss of blood. After an hour it rallied and apparently was in full posses-

sion of all its special senses, and was able to walk about as usual. The next day it manifested a ravenous appetite, and, during the whole time it was kept under observation, it showed no signs of illness or discomfort. The wound united by primary union, the skull showing the oblong bony defect at the site of the operation through which the pulsations of the brain could be distinctly seen and felt. Unfortunately the animal ran away after complete recovery had taken place and deprived me of the opportunity to study by post-mortem examination the local effect on the intracranial circulation by the operation. This experiment tends to prove that ligature of the longitudinal sinus can be performed without seriously compromising the functions of the brain, and that in certain well-defined instances this procedure might be resorted to in practice with a view of preventing or arresting hemorrhage from, and the entrance of air into, this vessel, in intentional or accidental wounds of the sinus.

Experiment No. 2.—Small tan cur, weight 10 pounds. Partial ether anæsthesia. Longitudinal sinus opened by two transverse incisions in close proximity; hemorrhage alarming, at first in jets, and, as the bleeding diminished, in a more continuous flow. At first the blood was bright red, but as respiration became impaired, it grew darker in color. Dilating forceps were introduced into the proximal wound, the hemorrhage continued, but no air entered as long as the animal was in a lying position, but as the respiration became more irregular and superficial artificial respiration was resorted to, and the head placed in an elevated position, whereupon the heart suddenly ceased to pulsate, and, upon applying the ear to the precordial region, a few irregular and very feeble contractions were heard, attended by a distinct churning sound, when the animal suddenly expired. Before death electricity was used with the effect of improving the respirations, but it had no effect whatever upon the action of the heart. Death took place about three-quarters of an hour after the sinus was opened. At the examination, immediately after death, all the tissues and organs were found in an exsanguinated condition. All the vessels leading to and from the heart were carefully tied, and the organ removed. On being placed in water it floated like a cork; the right auricle and ventricle were dilated, and on being opened under water bubbles of air and only a very slight amount of spumous blood escaped. The pulmonary

artery was also distended with air. The left ventricle was almost completely empty. In this instance the animal almost bled to death from the wounds in the longitudinal sinus, and yet no air entered, although the wound was kept patent with a pair of forceps. The entrance of air was caused by the elevation of the head and the forcible movements of the chest during the performance of artificial respiration. To judge from the amount of air found in the right side of the heart and its effects, the air must have entered quickly and in considerable quantity, distending at once the right side of the heart to such an extent as to paralyze the muscles of the heart in the diastole, after a few feeble attempts to force it from the right chambers. I believe, if the animal had been left in the lying position, and the head dependent, that death would have taken place from hemorrhage, as the blood which was draining through the sinus prevented the entrance of air, but as soon as the head was raised, the contents of the sinus by gravitation flowed towards the heart, and air entered with it to fill the vacuum which was being prepared by the diminished blood supply to the brain, and the acceleration of venous return, as well as the increased aspiration of the chest, which was brought about by the attempts at artificial respiration.

Experiment No. 3.—Horse, about 12 years old. Partial chloroform anæsthesia. Animal kept lying on the ground, head even with the body. Longitudinal sinus exposed for about two inches and incised longitudinally one inch Hemorrhage very profuse; blood at first bright red, gradually growing darker in color; double wave well marked. After about three quarts of blood had been lost, and the hemorrhage still continuing at the same rate, and not being readily controlled by the ordinary compression, it was decided to implant an aseptic sponge into the sinus. This was done, and the external wound united over it by the continuous suture. No air was seen to enter the wound, and auscultation over the heart revealed no abnormal sounds. During the operation of chiselling, the apices of the frontal sinuses were opened, which led to the fear that infection of the wound would subsequently take place from this source. This expectation was realized. The animal rallied soon after the operation, and appeared to be quite well for three days subsequently, grazing in the pasture with other horses. On the morning of the fourth day it was found dead. Examination of the cadaver showed

that the proximal end of the sinus was closed by a thrombus firmly adherent to the walls of the vessels and the implanted sponge, but about the distal end of the sponge, at a point which corresponded to the opening in the frontal sinuses, the brain and meninges showed all the appearances of acute septic inflammation. If infection had not taken place the aseptic sponge would have fulfilled all the purposes for which it was intended—arrest of hemorrhage and obliteration of the sinus. It seems to me that in cases of uncontrollable hemorrhage from accessible venous sinuses, the implantation of an aseptic sponge would prove a safe and efficient measure against hemorrhage, and would offer no obstacle against obtaining primary union and definitive closure of the vessel, as during the process of granulation the sponge disappears by absorption.

Experiment No. 4.—Horse, 14 years old, in good condition. This experiment was made for the purpose of confirming the suspicions already gained that the force of gravitation constitutes the most important factor in determining the admission of air into an open sinus of the dura mater; consequently no anæsthetic was given, but the animal was firmly held by a bit, and the operation was performed, without any difficulty, while the animal was in a standing position with the head elevated. With the trephine and chisel an oval opening, about two and a half inches in extent, was made over the longitudinal sinus. After all oozing had ceased, and the sinus being fully in view, its anterior wall was divided completely in a transverse direction. The edges of the wound immediately retracted, forming a diamond-shaped opening through which blood escaped in moderate force, but not nearly as copiously as on previous occasions when the animals were in the lying position. During the first inspiration after incision air entered with a loud gurgling or lapping sound, and on applying the ear over the apex of the heart a loud churning sound was heard synchronous with the movements of the heart. During expiration air-bubbles were seen to escape from the proximal end of the sinus. As soon as the head was depressed the hemorrhage greatly increased, but air never entered in this position; but as soon as the head was elevated, hemorrhage either ceased entirely or was at least greatly diminished, but air was sure to enter during inspiration. These manœuvres were repeated a number of times, and always with the same results. As the amount of air which was aspirated increased, the respirations

became more labored, and signs of cyanosis became apparent. An attempt was now made to close the wound in the sinus by sutures, and in this way arrest the hemorrhage. Three catgut sutures were passed through both edges of the wound, but on attempting to approximate its margins every one of them tore through the tissues before the parts were in apposition, proving conclusively that transverse wounds of the longitudinal sinus cannot be sutured, owing to the unyielding nature of the tissues. The external wound was completely closed by the continuous suture, and a firm graduated antiseptic compress controlled the bleeding. During the whole time of the operation, which lasted over an hour, some one of the bystanders listened to the heart's action, and the loud splashing or churning sounds were constantly heard. When the animal was released it commenced grazing in the pasture, and appeared as well as before the operation. The heart was examined at intervals of thirty minutes, and the abnormal sounds grew more feeble, and after an hour had entirely disappeared. The sound produced by the entering air, I have described as lapping, resembling very much the sound produced by the lapping of a dog or cat; the best possible word for this sound is the German expression "schluerfend." When air enters through a wound of the longitudinal sinus this sound is characteristic, and is always the same, and, in case the animal operated upon is a horse, it is sufficiently loud to be heard at some distance. Experiments have shown that horses are most tolerant to the presence of air in veins, on account of the unusual development of the right ventricle, which has sufficient power to force the air through the pulmonary circulation, and this experiment would certainly tend to corroborate this observation, as air in large quantities was aspirated at least a dozen times during the operation, and that most of it entered the right side of the heart, and was not returned is evident from the persistence of the sounds, due to the presence of air for a period of two hours, and yet, aside from a certain degree of embarrassment of respiration, the animal suffered no inconvenience. The wound healed by primary union. The defect in the skull remained permanent. The animal was killed about four weeks afterwards. Post-mortem appearances: The trephine opening filled with cicatricial tissue. Proximal end of sinus, just behind trephine opening, contains one large granulation thrombus. Cicatricial tissue filling almost the entire lumen of the sinus. Anteriorly the sinus is somewhat contracted and smooth; no

thrombus here or evidences of proliferation. The circulation is apparently restored by the formation of a new channel or dilatation of a pre-existing one; this new sinus is located to the left of the median line. The lateral sinuses are very much enlarged.

Experiment No. 5.—Young yellow dog, weight about 15 pounds. Partial ether anæsthesia. Longitudinal sinus exposed and transversely incised at two points in close proximity. Hemorrhage profuse, was allowed to continue for over half an hour in order to estimate the length of time which would be necessary for death to occur from this cause uncomplicated by admission of air. When the animal appeared moribund both ends of the sinus were ligated. The heart's action was very feeble and irregular while respiration was entirely suspended. Artificial respiration was kept up until the heart's action ceased. Death occurred in thirty-five minutes. At the examination after death no air was found in the vessels or heart, and death was plainly attributed in the absence of any other cause solely to the loss of blood.

Experiment No. 6.—Newfoundland dog, weight 50 pounds. Partial ether narcosis. During the removal of bone over the sinus severe hemorrhage was encountered from the large venous channels in the diploë. The great irregularity of the external surface of the skull led to a mistake, as the frontal sinuses were again opened. The longitudinal sinus was laid open by an incision half an inch in length in a parallel direction to the vessel. Hemorrhage very profuse for half an hour, checked at times by compression, when it finally diminished and the wound was closed. After the operation the animal walked with a staggering gait, and would run against objects indiscriminately, showing that sight was greatly impaired, inasmuch as the animal had fully recovered from the effects of the ether. No air was seen or heard to enter the sinus. Heart sounds feeble, but otherwise normal. Death in this case took place a week after the operation from lepto-meningitis. The source of infection undoubtedly was again traceable to injury of the frontal sinus, as the earliest evidences of the disease were found nearest the opening in this structure.

Experiment No. 7.—Old decrepit horse. Operation was performed while the animal was in the erect position. On removing

disk with trephine a longitudinal wound one-half inch in length was found in the anterior wall of the sinus through which bright red blood escaped. Double pulsation well marked. Almost immediately after the removal of the disk of bone, and before more than an ounce of blood had escaped, air entered with a loud and distinct lapping sound, audible to all who were present. On applying the ear over the heart the same loud churning sounds were heard. As the head was lowered the flow of blood became more forcible and copious, but no air entered; as soon, however, as the head was elevated, bleeding diminished, and air entered during almost every inspiration. Respiration became labored, and after air had entered four or five different times in succession the animal fell to the ground. In this position no further entrance of air occurred, but the hemorrhage continued copiously, the blood flowing in a continuous stream with a well-marked double jet synchronous with the action of the heart and the respiratory movements of the chest. The opening in the skull was enlarged to two inches in length and one and one-half inches in width, so as to expose the sinus freely. A number of catgut sutures were now introduced through both lips of the wound, and on attempting to tie them great difficulty was experienced in approximating its margins, which could be brought nearly but not completely in contact without the sutures tearing through. The tying of all the sutures resulted in diminishing but not arresting the bleeding, showing conclusively that in longitudinal wounds of the sinus suturing is an imperfect and unreliable measure in arresting hemorrhage, to say nothing of the difficulty which is experienced in passing the sutures at such great depths and in the limited space furnished by the artificial opening in the skull. The wound was not closed but tamponed with iodoform cotton in order to observe from time to time the processes which nature would initiate in the restoration of the wounded sinus. Half an hour after the first entrance of air the churning sounds in the heart had much diminished, and almost completely disappeared after the lapse of one hour. The animal recovered completely from the immediate effects of the air-embolism, the respiration having again become normal in frequency and character. After two hours the tampon was removed, but bleeding again occurred, and it was replaced. The animal died twenty-four hours after the operation, probably from the combined effect of loss of blood, hemorrhage into the subdural spaces, air-

embolism, and senile marasmus. At the examination of the cadaver a subdural clot was found on the right side of the brain which weighed about half an ounce, on the left side of the sinus a second but smaller subdural clot was found. Trephine opening filled by a coagulum. One of the sutures had lost its hold by tearing through the tissues of one margin of the wound. Within the sinus a small fragile clot was found in the lateral wall of the sinus which served as the source of hemorrhage into the subdural space. This experiment illustrates well the danger of plugging the opening in the skull for the purpose of arresting hemorrhage in case the lateral walls of the sinus are injured, as it will almost necessarily lead to subdural hemorrhage and expose the patient to all the disastrous consequences incident to this occurrence. In this instance sponge implantation would not only have more successfully guarded against external bleeding, but would also have served as a sure prophylactic against extravasation into the subdural space. It also teaches that suturing in cases of wounds of the longitudinal sinus with limited defects in the bony walls of the cranium is impracticable, unreliable, and unsafe, and should never be resorted to unless the dura mater is so extensively exposed or separated as to permit, by making gentle traction, perfect and complete approximation of the margins of the wound.

VII. *Practical Suggestions.*

In order to study the conditions which favor the aspiration of air into a wounded sinus of the brain it is necessary to call attention to some of the peculiarities of the intracranial circulation. Mosso,[1] who has made this a special subject of investigation, asserts that the intravascular presence in the veins within the cranium is higher than in the veins of any other part of the body. Actual measurements have shown that the blood-pressure in the longitudinal sinus is equal to 100–110 mm. Hg. The probable cause of this phenomenon is that the force of distension of the arteries within the closed and unyielding cranial cavity is added to the *vis a tergo*. The intracranial veins show distinct pulsations which are dependent upon the pulsations of the arteries, and their movements are so plain that they can be

[1] Ueber den Kreislauf des Blutes im menschlichen Gehirn, 1881.

graphically demonstrated ; every diastolic movement in the artery corresponds to a venous pulse. During the pulsations of the brain, Mosso claims with Donders and Berlin, that the cerebro-spinal fluid does not escape into the spinal canal. In a case of spina bifida he has been able to trace respiratory but no circulatory movements. When the tumor was compressed only a very slight increase in the volume of the brain could be detected at the fontanelle, even if nearly the whole contents of the tumor were pressed into the spinal canal. G. Burkhart[1] has published the results of his observations on the movements of the brain which he made on four patients who had suffered partial loss of the cranial vault. The tracings obtained represented three forms of movements—pulsatile, respiratory, and vascular. The cerebral pulsation has the form of a tricrotic or tetracrotic pulse, the phases following one another in about the same time as those of the carotid pulse. His observations lead him to the conclusion that the brain presents the same movements within the intact skull as in the infant or when a defect in the skull exists, the result of traumatism. The brain expansion is synchronous with the dilatation of the vessels and takes place in the direction of the vascular ramifications. The resistance is in the inverse proportion to this expansion. In the closed skull the excess of pressure in the arteries aids materially the propulsion of the blood through the veins, and also that of the serolymphatic fluid. In the open skull the curve rises during expiration and falls during inspiration. All actions which increase the respiratory movements increase the height of the curve. A secondary elevation follows labored inspiratory movements, but the pulse waves are never completely effaced. The vascular curves occur independently of respiration or pulsation. The height of the curves bears no constant relation to their length. They are notably influenced by psychical influences. They are produced by movements of the vessels by means of the vasomotor nerves, and can be made very conspicuous by irritation of the cervical sympathetic. Bergmann,[2] in his remarks on the

[1] London Lancet, Oct. 15, 1881.

[2] Verhandlungen d. deutschen Gesellschaft f. Chirurgie, vol. x. p. 14.

movements of the brain (in opposition to Mosso) at the meeting of the Congress of German Surgeons in 1881, insists that the cerebro-spinal fluid acts as a regulator in maintaining the equilibrium between the arterial and venous circulation within the cranium. The pulsations of the sinuses of the dura mater were discovered and studied under his supervision at Dorpat as early as 1873. He argues that these pulsations are very slight, and on that account insufficient to counterbalance the arterial pulsations. He explains the pulsations of the sinuses in the same way as Donders and Jacobi have accounted for the pulsations in the veins of the papilla of the optic nerve. The pulsations are the result of increased tension in the cerebro-spinal fluid during the arterial systole and the consecutive diminution of intracranial pressure during the arterial diastole. The cranium being a closed cavity with unyielding walls, it is not difficult to understand that in case one of the sinuses is opened by a wound which communicates with the atmospheric air, the sudden loss of blood will have a tendency to create a vacuum which is filled by the admission of air which reaches the left side of the heart with the venous blood. All circumstances which diminish intravascular and intracranial pressure must of necessity favor the occurrence of aspiration of air into a wounded sinus. It is evident that aspiration of air into an open wound of the longitudinal or any other sinus of the dura mater is favored by the following conditions: 1. The force of gravitation. 2. The inspiratory movements of the chest. 3. The condition of arterial circulation. In considering the prophylactic treatment against the admission of air during operations which involve any of the cerebral sinuses, it is of the greatest importance to keep the head at a level with the heart to insure regular respiration and to guard against undue or forcible inspiration, and, finally, to maintain the normal activity of the ventricular contractions. The direct preventive measures consist in:—

1. Continuous irrigation of the field of operation. 2. Prophylactic ligation of the sinus.

In resorting to constant irrigation the fluid used should be an aseptic solution at the temperature of the body, which, if it

should enter the venous circulation to fill an empty space, would do no harm either as a toxic agent or by causing coagulation of the blood. A solution of salicylic acid in distilled water, or borated water would be best adapted for this purpose. In extirpating tumors of the dura mater in the region of the longitudinal sinus, where wounding of this structure becomes a necessity, it would not only be prudent but good practice to ligate the sinus on each side before attempting the removal of the tumor, as this precaution would surely and effectually prevent the two most dangerous and alarming accidents—hemorrhage and aspiration of air. This plan was followed by Kuester in removing a sarcoma of the dura mater in 1881.[1] Experiments on animals and the cadaver have convinced me that this operation can be performed with comparative ease, if the defect in the skull is sufficient in extent to permit the necessary manipulations, and, in the event this operation be done for the purpose of facilitating the removal of tumors of the dura mater, this precaution should never be neglected. With a tenaculum the dura mater is seized and drawn forward at the outer border of the longitudinal sinus, and a small incision parallel with the border of the sinus should be made with a tenotome, the incision being only sufficiently deep to divide the dura mater. After making such an incision on each side of the sinus directly opposite each other, the sinus should be grasped with a sharp-toothed spring forceps and drawn forward, when a small curved sharp-pointed aneurism needle is passed into one of the openings, and, after penetrating the falx cerebri underneath the vessel, is brought out through the opening on the opposite side. When both of the ligatures are in place the peripheral ligature is tied first, and after emptying the intervening part of the vessel of its contents the proximal ligature is also tied. If both of the ligatures have been properly applied the intervening portion of the sinus can be opened or excised with the tumor without risk of hemorrhage or the introduction of air, at the same time it will greatly facilitate thoroughness in the removal of diseased tissue. I am firmly

[1] Berl. Klin. Wochenschrift, p. 673, 1881.

convinced that the preliminary ligation of the longitudinal sinus will become an established procedure in all cases where tumors of the dura mater are so situated that this removal implicates this structure and that it will render possible the removal of tumors which without it would place in great and immediate jeopardy the life of the patient by hemorrhage or the admission of air. The process of cicatrization in the sinus is the same as in veins, and is accomplished in the same brief period of time. In accidental wounds of the sinus ligation should be resorted to whenever the original defect in the skull is sufficient to permit the necessary manipulations or when simpler measures have failed to accomplish the same object. Implantation of an aseptic sponge into a wounded sinus should be resorted to in all cases of wounds of the lateral walls of the sinus, in cases of accidental wounds where ligation is impossible and where other measures have failed to arrest the hemorrhage. The sponge should be large enough to make gentle pressure upon the inner surfaces of the sinus, and yet sufficiently firm to arrest the circulation in the vessel so as to prevent the escape of blood into the subdural space. The hæmostatic action of the aseptic tampon is made more efficient by adding external compression, applied in the form of a graduated aseptic tampon. If the wound remains aseptic the sponge forms a nucleus for the thrombus and is infiltrated by connective-tissue cells from the intima and adjacent tissues, and is gradually removed by absorption as the definitive obliteration of the vessel proceeds. Small wounds of the sinus can be readily and safely closed with the lateral ligature applied in the same manner as in similar wounds of the veins.

In recapitulation we are warranted in stating the following conclusions :—

1. Elevation of the head is the direct and most essential cause in the production of air-embolism through a wound of the superior longitudinal sinus.

2. Suturing of a wound of the superior longitudinal sinus as a hæmostatic procedure is unreliable, and in most instances anatomically impossible.

3. Prophylactic ligation of the superior longitudinal sinus

should be resorted to in all cases where this vessel is involved in extirpating tumors of the dura mater.

4. Implantation of an aseptic sponge into a wounded longitudinal sinus will arrest hemorrhage without interfering with the definitive obliteration of the vessel, and deserves a trial in cases where the lateral walls of the sinus have suffered injury and where ligation is impracticable.

VIII. *Immediate Cause of Death after Intravenous Insufflation of Air.*

Various theories have been advanced to explain the injurious effect of the presence of air in the circulation. Bichât[1] attributed death resulting from intravenous injection of air to cerebral anæmia produced by the presence of air in the cerebral vessels, asserting at the same time that a very small quantity would suffice to produce this effect. As the first argument in favor of this view, he claims that the heart continues to beat for some time after the cessation of animal life. Secondly, air injected through one of the carotids produces death in the same way as when introduced into the veins. Thirdly, the cases reported by Morgagni, where death was attributed to the presence of air which was found in the cerebral vessels at the post-mortem examination, and which was supposed to have developed there spontaneously. Fourthly, all examinations after death revealed the presence of frothy blood, mixed with air-bubbles in both ventricles. Fifthly, air injected into one of the divisions of the portal vein produces no ill-effects until it reaches the general circulation. Sixthly, the almost instantaneous death observed in some instances is due to the acceleration of the heart's action, and consequently the rapid conveyance of air into the cerebral vessels. Seventhly, the existence of convulsions which he ascribes to the irritating quality of the air on the brain. He summarizes as follows: "We shall conclude that in the accidental mixture of air with the blood of the venous system, it is

[1] Physiological Researches on Life and Death, p. 186.

the brain which dies the first, and that the death of the heart is the consequence of the death of the brain."

Magendie, in commenting on Bichât's views concerning the manner of death from intravenous injection of air, remarks: "This is not correct, and death takes place, on the contrary, by the cessation of the motions of the heart. The right ventricle is filled with air, and this air, dilated by heat, so distends it that it can no longer contract." Magendie also claimed that small quantities of air in veins will not result in death, and that life is in jeopardy only when the air is injected suddenly and in considerable quantity. He relates the details of an experiment which he made on a horse, where he injected in rapid succession into the veins of the animal forty syringefuls of air, and three syringefuls into the carotid artery. The capacity of the syringe was seventeen centilitres. The animal died three minutes after the last injection. At the examination of the body he found air in the azygos vein and in the thoracic duct which contained much lymph, as well as in the lymphatic vessels of the internal surface of the lungs. The heart was enormously distended with air mixed with a small quantity of blood. Morgagni, Brunner, Sprœgel, and Nysten referred the cause of death to the same source—over-distension and paralysis of the heart. When death is not produced by the mechanical effect of the air on the heart, then the consecutive symptoms were referred by Nysten to an obstruction of the lungs produced by the accumulation of air in the ultimate divisions of the pulmonary artery. He observed that the embarrassment in respiration often appears as late as twelve hours after the introduction of the air, and becomes greater and greater; the bronchi are filled with a viscid fluid, and the animal usually dies on the third or fourth day. In such instances no air was found in the heart or the vessels, but the lungs, instead of being pink-colored, were grayish, tinged with brown, and loaded with frothy blood and mucus. The same views were entertained by Boerhave, Kettler, and Beck. In such cases death results from asphyxia from the mechanical obstruction to the passage of the venous blood through the pulmonary circulation. Mery accepts the views of Mercier

published in 1839, who attributed death to this, that the blood mixed with air becomes frothy, enters the pulmonary capillaries and obstructs them. Physicists are aware that a capillary tube which readily admits the passage of air or water offers a great resistance to a mixture of air and water; this mixture causes a series of bubbles, separated by minute septa of liquid. Poisenille has shown by a series of experiments that such in fact is the cause of death whenever air mixed with blood has obstructed the pulmonary capillaries. The right heart remains over-distended and cannot be emptied.

Blundel[1] studied the effects of the intravenous injection of air in his experiments on transfusion of blood. He injected five drachms of atmospheric air into the femoral vein of a small dog in quantities of a drachm each at a time, without any serious effects. The symptoms observed were sighing respiration, irregular pulse, muscular tremors, and vomiting, all of which, however, subsided after a brief space of time, and the animal recovered completely in three days. A second experiment was made on the same animal by blowing three drachms of pulmonary air into the femoral vein without even producing much *temporary* inconvenience. He concluded that it seemed indisputable that small quantities of air may be introduced into the circulation without destroying life. Dr. Haighton made the same experiments with intravenous injection of air and with like results. A series of experiments were made by Panum.[2] The subject of the first experiment was a small dog in which five cubic centimetres of air were injected into the lower portion of the jugular vein. No symptoms followed immediately after the operation. Four days subsequently the weight of the animal was reduced from 3540 to 3030 grammes. The tissues around the point of puncture were inflamed. On the fifth day the animal was killed. The skin of the animal was dotted with spots of ecchymosis resembling the extravasations as they occur in the disease known as morbus macu-

[1] Medico-Chir. Trans., vol. ix. p. 65.

[2] Experimentelle Beiträge zur Lehre von der Embolie. Virchow's Archiv, vol. v. p. 499.

losus Werlhofii. In the vicinity of the wound, the tissues were emphysematous, emitting an offensive odor. Among other post-mortem appearances the lungs presented several gray superficial stripes and spots, 1–2 lines in diameter. In the middle of several of these patches were found empty spaces which Panum regarded as encysted air-bubbles. In addition small isolated nodules were also found which contained in their interior small bubbles of air. These nodules contained also numerous nuclei and fat globules. In the second experiment ten cubic centimetres of air were injected into the lower portion of the jugular vein of a small one-year old dog. During the first three days nothing was observed with the exception of rapid emaciation. On the fourth day the animal began to lose the hair not only in the vicinity of the wound, as in the first case, but over the entire surface of the body. Later inflammation and ulceration attacked the point of operation. On the eleventh day the animal was killed. The lungs again presented subpleural nodules, the size of a pin's head to that of a grain of sand, in which could be found large cells filled with fat molecules, granular cells, and fat globules in large number. Air could not be found in any of them, and their relation to the capillaries of the pulmonary artery could not be determined. In the third experiment 30 cubic centimetres of air were injected into the jugular vein without producing any alarming symptoms, except rapid and deep respiration. The next day no change was observed, but on the following day the animal was found dead. A careful examination revealed punctiform extravasations in different portions of the brain, stomach, and liver. The wound showed a healthy appearance. The lungs contained many very small yellowish-white nodules and points of extravasation one to two lines in diameter. The hemorrhagic infarcts contained each a minute cavity filled with air. The nodules also contained air. From these experiments it appears that the air-emboli which passed through the pulmonary capillaries produced local disturbances in the minute vessels in the skin and gastro-intestinal canal, while the air-emboli in the ultimate branches of the pulmonary artery gave rise to circumscribed foci of inflammation.

Picard[1] found after insufflation of air into the portal vein intense hyperæmia in the distal portion and radicles of the vessel, the same as after ligation. At the commencement of the insufflation the blood pressure in the femoral artery and one of the rectal veins was about the same, but after a while the pressure in both was simultaneously diminished, but the positive pressure lasted longer in the vein than in the artery. The action of the heart was increased, the respirations became slower, and the temperature in the rectum was gradually reduced. As an interesting physiological fact it is mentioned that after insufflation of air into the portal vein no sugar could be found in the liver, and the fibrin in the portal blood was diminished.

The experiments of Magendie, Bouillaud, and later those of Couty,[2] prove conclusively that in cases of entrance of air into a peripheral vein the air collects almost exclusively in the right side of the heart; only a minute quantity entering the left side through the pulmonary capillaries. Referring to this question Flint says: "The production of death from air in the veins is purely mechanical. The air, finding its way to the right ventricle, is mixed with the blood in the form of minute bubbles and passed into the pulmonary artery. Once in this vessel, it is impossible for it to pass through the capillaries of the lungs, and death by suffocation is the inevitable result if the quantity of air be large. It is because no blood can pass through the lungs that the left cavities of the heart are usually found empty."[3] If the quantity of air introduced is small, or the entrance repeated in small quantities, the air collects in the capillaries of the pulmonary artery as air-emboli, obstructing the circulation in the impacted vessels, but a sufficient number of vessels remain pervious to maintain the circulation, and life is prolonged until the equilibrium of the circulation is restored by the absorption of the adventitious air. No traces of air are found in the arteries, only a small quantity passing into the venæ cavæ through the tricuspid valve, which has been rendered insufficient by the

1 Sur les injections d'air dans la veine porte. Gaz. Méd. de Paris, No. 6, 1873.

2 Étude experimentale sur l'entrée de l'air dans les veines, Paris, 1875.

3 Physiology of Man, Blood, Circulation, Respiration, p. 323, 1866.

presence of air. When a considerable amount of air has entered the right ventricle of the heart mixed with the blood, the air is separated from it, and, its specific gravity being less, it rises to the highest point of the chamber in contact with the anterior walls; besides it expands and by this over-distension it impairs the muscular contractility which even in a normal condition is insufficient to empty the cavity completely. The impediment to the circulation being the presence of the large air-embolus in the right ventricle, which on account of the higher temperature increases in volume, sudden death will take place in the diastole by an arrest of the heart's action from paralysis by over-distension. If the animal escapes instantaneous death from this cause, the heart is inadequate to force the blood from the right ventricle through the pulmonary circulation, as its efforts are expended in compressing the air; only a minute quantity of blood being forced into the lungs. In proportion as the amount of air and blood in the right ventricle increases, the right side of the heart is expanded and the volume of blood in the lungs and the left heart is diminished. At last the distended walls of the heart prevent perfect closure of the tricuspid valve, giving rise to venous pulsation, a constant symptom in all cases of air-embolism which prove rapidly fatal. On the advent of this complication the intra-arterial pressure in the pulmonary and peripheral arteries diminishes, which further enfeebles the pulmonary circulation, at the same time it produces acute anæmia of the brain, and death results from anæmia of the brain or asphyxia. Such is the mechanism of death in cases of entrance of air into the venous circulation, and at the same time it offers an explanation why in these cases the air is found mostly in the right side of the heart and the large venous trunks. These facts also corroborate the observations of experimenters that certain animals succumb more readily to the presence of air in veins than others. The tolerance of intravenous air is most marked in animals with well-developed respiratory organs and a proportionately powerful right ventricle. In horses, for example, the volume of the right cavities is smaller and the muscular structure more powerful, circumstances which

explain the fact that entrance of air into the veins of these animals does not easily kill them. The circulatory apparatus of the dog offers the least resistance to intravenous air. Rey blew air into the jugular veins of horses after venesection without producing death. Small amounts of air produced no result whatever, and a volume of air equivalent to two expirations proved fatal only in debilitated animals. Some animals remained well after leaving a canula in the jugular vein for several hours. Death was sure to follow if after the insufflation of air the vein was ligated. Animals who have previously lost large quantities of blood readily succumb to intravenous admission of air.

Laborde and Muron, who studied the effects of intravenous and intra-arterial insufflation of air, placed great stress on the manner in which the injections are made in determining the gravity of the symptoms. They observed that in case the insufflations into veins are made slowly and repeated at intervals large quantities were tolerated without exciting serious symptoms. If, on the other hand, they were made suddenly and with force, and the quantity of air was considerable, death resulted almost instantaneously from arrest of the heart's action in the diastole, an occurrence which was attributed by these authors to over-distension of the right side of the heart.

In repetition, it may be stated, that the immediate cause of death after intravenous injection of air has been referred by different experimenters to :—

1. Mechanical over-distension of the right ventricle of the heart and paralysis of this organ during the diastole.
2. Acute cerebral ischæmia.
3. Asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery.

IX. *Intra-Arterial Insufflation of Air.*

As the accidental admission of air never takes place in wounds of the arteries on account of the high degree of intravascular pressure and the absence of any aspiratory force it is not sur-

prising that the effect of the artificial introduction of air into these vessels has been made less frequently an object of experimental research than intravenous insufflation of air. The subject is devoid of any practical value, and the interest attached to it is of a purely scientific nature. As we have already seen, Bichât entertained the idea that atmospheric air acts as a direct irritant to the substance of the brain when brought in contact with that organ through the medium of the cerebral vessels, and death from insufflation of air, whether into veins or arteries, was invariably attributed by him to cerebral anæmia. Panum,[1] in his researches on embolism, made arterial insufflation of air also a subject of experimentation. A rapidly fatal termination followed the injection of 4 c. centimetres of air into the lower portion of the carotid artery of a medium-sized dog. The infundation was superseded immediately by severe general convulsions, alternated by violent attacks of rage. The animal foamed at the mouth, and had involuntary discharges from the bladder and bowels, the eyes became prominent, and the pupils, at first dilated, contracted, and remained so permanently. The animal soon became quiet and motionless. Respiration and heart's action slow. The right anterior and both posterior extremities were extended and rigid, while the left front leg remained relaxed. Conjunctiva insensible, while touching the cornea produced contractions of the eyelids. For two hours the animal remained in a condition resembling anæsthesia without changing its position, with slow pulse and respiration, when life ceased without a tremor or convulsion. The post-mortem examination showed numerous punctiform ecchymoses in the gastro-intestinal mucous membrane, the liver, diaphragm, and abdominal muscles. The superficial vessels of the brain, more particularly the veins, were extremely hyperæmic; the jugular veins were distended to their utmost with blood. The small arteries contained many air-bubbles, so that the smallest vessels presented varicose dilatations which resembled a string of pearls. The large vessels at the base of the brain also contained air, and

[1] Op. cit.

numerous red spots were disseminated throughout the white substance of the brain.

Laborde and Muron[1] witnessed after the introduction of 20–60 c. cm. of air into the carotid artery of dogs, when injected in a peripheral direction, that death was produced rapidly and was always preceded by tetanic convulsions and labored respiration, while if the quantity of air injected was smaller, and especially if the air was thrown in in divided doses, the animal often survived the experiment for twenty-four hours. In the latter class of cases the operation was followed by tetanus, vomiting, paralysis, and coma. The autopsy revealed softening of the brain, and capillary hemorrhages, especially in the middle portion of the brain, the medulla oblongata, and in the posterior lobes of the cerebrum. The intra-arterial insufflations of air in all these experiments were immediately followed by grave cerebral symptoms which can only be interpreted by the constant post-mortem appearances, air-embolism of the cerebral vessels, and extreme ischæmia of the brain. The presence of air in the arteries, on the left side of the heart, is followed by an entirely different series of phenomena than in the veins on the right side of the heart. The acute ischæmia of the brain thus induced is invariably manifested by tetanic rigidity of the voluntary muscles and almost complete suspension of the respiratory movements of the chest. The contractions of the left ventricle are so powerful as to overcome these additional impediments and to completely evacuate the chamber. The air is expelled as soon as it enters and is distributed throughout the whole arterial system. It must be readily conceived that the air when it has reached the aorta will rise into the carotid arteries and thence into the cerebral vessels, distending them to their utmost capacity. In such instances death results from sudden cerebral anæmia before the air can gain entrance into the venous system through the capillaries. That the presence of air is detrimental to the nervous system has been established by experiments on animals. Bohnius attributed to the air when introduced into the vascular

[1] Virchow u. Hirsch's Jahresbericht, vol i. p. 268, 1873.

system poisonous properties, and this opinion is entertained by Neudörfer even at the present time. Copeland believed that the oxygen of the adventitious air combines with the carbonic oxide in the venous blood producing carbonic acid. These and similar theories do not explain the phenomena observed after insufflation of air. The symptoms during life, and post-mortem appearances point directly towards physical obstruction in the bloodvessels by air-emboli which suspends the function of one of the vital organs, and consequently must be regarded as the immediate or direct cause of death. Insufflation of air into the carotid artery towards the brain is almost immediately fatal. If a moderate quantity of air is forced through the arterial system into any other organ except the brain, except by forming a temporary obstruction in the circulation, it does no particular harm and is in a short time removed by absorption. This fact may have induced Flint to make the following remarks concerning this subject:[1] "Air injected into the arteries produces no such serious effects as air in the veins. It is arrested in the capillaries of certain parts, and in the course of time is absorbed without having produced any injury."

The danger arising from the introduction of air into the arteries or left side of the heart arises from obstruction to the circulation through the cerebral vessels by air emboli and the cessation of the functions of the brain consequent upon an almost complete ischæmia of this organ. The mechanical distension of the left side of the heart by the accumulated air is overbalanced by the powerful contractions of the left ventricle, which are sufficient to empty the chamber almost completely, and to force the air into the ultimate distributions of the arteries causing acute anæmia in distant organs, but more particularly in the brain. In these instances the air is forced directly into the cerebral vessels either by the injecting force or the powerful contractions of the left ventricle, and if death takes place it follows as the direct result of acute cerebral ischæmia.

[1] Op. cit., p. 324.

X. *Clinical Study of Air-Embolism.*

All physiologists agree that regular and deep inspirations produce a powerful aspiration in the large veins near the base of the heart, more particularly in the jugular and subclavian veins. This fact is so well known that the base of the neck and the clavicular regions are frequently referred to as the "danger zone." During the inspiratory act the chest expands, and the flow of venous blood is accelerated towards the cardiac or proximal side. A diversity of opinion still exists among authors in regard to the distance to which this force directly affects the venous circulation. Experiment and clinical observation have shown that the danger of entrance of air into wounded veins is increased as the wound approaches the heart. Berard studied the anatomical conditions of the vessels where entrance of air has most frequently taken place, and he came to the conclusion that this accident can only happen in the event when the wounded vessel is empty, and its walls are prevented from collapsing, and the wound remains patulous. He found these conditions normally present in the sinuses of the dura mater, the hepatic veins, the superior vena cava, the internal jugular, the subclavian, and axillary veins, because the walls of all these vessels are firmly fixed to the adjacent tissues, which prevents their collapse on being wounded. It has been further shown that the admission of air is favored by pathological conditions which affect the veins in a similar manner, as the existence of induration of their walls, the result of chronic inflammation, or infiltration by neoplasms. Experience has corroborated these views, inasmuch as it has been shown that this accident has occurred most frequently in operations in the vicinity of veins which, from their anatomical location, are prevented from collapsing by firm and unyielding layers of fascia, and vessels which are, or have become, adherent to unyielding tissues. Again, it is always very justly feared in removing tumors which have become adherent to large veins, as the morbid process frequently has impaired the normal resiliency of the vessel so as to keep its lumen patent in the event it is wounded during the dissection.

In 33 cases of intravenous aspiration of air collected by Couty,[1] air entered the external jugular 9 times, the axillary 8 times, the internal jugular 5 times, the subscapularis 3 times, the facial twice, the anterior jugular twice, the occipital twice, and twice one of the anterior thoracic veins in close proximity to the clavicle. In 1864 Greene collected 67 cases where air had entered a vein during an operation. The greater number of cases occurred during extirpation of tumors in the region of the neck, chest, and axilla. Twice the accident took place in disarticulating the humerus at the shoulder-joint (Cooper, Delpech); once on extirpating the scapula and clavicle (Mussey); twice on tying the subclavian artery (Rigaud, Clemot); three times on bleeding from the external jugular vein, three times on bleeding from the median vein, and once on passing a seton-needle through the tissues in the regions of the neck. Among the wounded veins the external jugular is mentioned 13 times, the internal jugular 10 times, the subclavian and axillary each once. In the remaining cases the vein is not specified, or the injury involved a branch in close proximity to the specified vessels.[2] All causes which interfere with the free return of venous blood prevent the admission of air; while, on the other hand, all influences which promote the venous circulation, such as an unimpaired *vis a tergo*, regular deep inspiration, and the force of gravitation predispose to this accident. Soon after Beauchène made known his case the Royal Academy of Medicine of Paris appointed a commission to investigate the subject. In the report it is stated that the results of the experiments, which were made principally on dogs, had proven that the conditions necessary to determine the entrance of air consisted in making the wound in the vein anywhere within the area of the venous pulse, or, at any rate, only a short distance from it. If the wound was located at a greater distance and beyond the influence of the venous pulsations, no air would enter, although the wound was kept open. The experiments were also to determine the extent of the venous pulse, and the conclusions arrived

[1] Op. cit.

[2] American Journal of the Medical Sciences, 1864, p. 38.

at were that the brachial and axillary veins were beyond the venous wave, while the subclavian and lower third of the jugular veins were the seat of pulsations, consequently wounds of the veins in the localities were liable to admit air. The sound produced by the entrance of air is described as resembling the lapping of a dog or cat, and that it always occurred during, and synchronous with, inspiration; but sometimes, when it was heard more frequently, it accompanied the diastole of the right ventricle. After the air had entered the vein, the sound which could be heard on auscultation over the heart was described as a "bruit de soufflet," synchronous with the action of the heart. In regard to the effect of the aspirated air it was decided that, in order to produce a fatal result, it was not only necessary that the amount of air introduced should be considerable, but that it must be thrown into the vein with some degree of force.

Hertwig called special attention to the fact that aspiration of air is not as frequent an accident as is generally supposed, and that for its occurrence it is necessary that the peripheral flow of blood to the wound must be obstructed, that the edges of the vein wound must be drawn apart, and finally, that even the introduction of a canula into the vein is necessary to admit a sufficient amount of air to produce serious results.

The first case of admission of air into a vein that was recognized and verified by a post-mortem examination occurred in the practice of Beauchêne, and is described by F. Magendie.[1] As the case is of great historical and scientific interest, I will relate it as described by Magendie.

A locksmith, 23 years of age, had for five years a large tumor on the right shoulder and clavicle. His acute sufferings induced him to enter the hospital to have it removed. It was necessary in the operation to remove the middle portion of the clavicle. Thus far the success was complete; but little blood was lost, the pulse was good, and the breathing easy, when the patient suddenly cried out: "My blood is leaving my body! I am dead!" At the same

1 Physiological Researches on Life and Death, by X. Bichât, with Notes by F. Magendie. Translated. Boston, 1827, p. 188.

moment he became stiff, lost his consciousness, and was covered with a cold sweat. A singular and rather loud noise was heard in the interior of his chest. The surgeon thought that he had opened the pleura by removing a portion of the clavicula, and thus given access to the air and to the blood to the right side of the thorax. The fingers of an assistant were immediately thrust into the bottom of the wound with a view of stopping the supposed opening in the pleura, and the surgeon endeavored to introduce into the thorax the extremity of a sound of gum-elastic. When he thought that he had succeeded he drew with his mouth the air which he supposed to be effused in the pleura. He wished then to proceed to the dressing, and, in order to do this, he substituted for the fingers of the pupil, which were at the bottom of the wound, a sponge covered with wax; but the moment the sponge took the place of the fingers, the same noise that was at first heard, and which had ceased in an instant, was renewed with more force than before. The syncope and cold sweat still continued. Water thrown into the face made him give some signs of life, but he died a quarter of an hour after the appearance of the accident I have first described, and forty-five minutes after the commencement of the operation. The body was examined the next morning. They expected to find the right pleura open, much blood and air effused into its cavity, and the lungs on that side collapsed. Nothing of the kind was found. The pleura was whole and there was no effusion in it. The lungs were as usual, but an opening of half an inch in extent was discovered in the external jugular vein, at the place where this vein opens into the subclavian. The cavities of the heart were large, but contained no blood. Bubbles of air were observed in the vessels of the brain; the other vessels were not examined.

In order to study some of the conditions under which air has been aspirated into veins, and for the purpose of ascertaining the effects of such accidents in man, I will introduce a number of well-authenticated cases which will represent a great diversity in the point of entrance, and will also aid in the establishment of the fact that this accident can occur outside of the regions of venous pulse, and always occurs during inspiration, and is never produced by the aspiratory function of the heart.

External Jugular. Barlow's case.[1] The patient was a female suffering from a tumor seated on the side of the neck, which had been increasing in size for several years; its base was extensive, and occupied the whole of the lateral and posterior parts, extending from the ear to near the sternum, and sidewise from the thyroid gland to the sterno-mastoid muscle, under which a part of the tumor was situated. The patient was seated in a reclined chair, supported by assistants. Two superficial elliptical incisions, ten inches in length, were made downwards from a little below the ear, "when on proceeding to dissect the skin aside to get at the basis of the tumor, a sudden and unexpected hissing and gurgling noise rushed obviously from a large divided empty vein, and the patient expired instantly, without either sigh, groan, or struggle, and every effort to restore animation became fruitless." The divided vein appeared larger than the normal external jugular, but the reporter believes that it was this vessel or an anomalous vessel greatly enlarged. As the incisions must have traversed the external jugular, according to his own description, it was undoubtedly this vessel which was injured. It is distinctly stated that the vessel was flabby and empty, and that the instant the atmospheric air gained access and filled the vacuum, the hissing noise ceased, the patient expired, and the mouth of the vessel collapsed.

Remarks.—In this case the admission of air was favored by the dilatation of the vein, and the semi-erect position of the patient. The latter factor produced the emptiness of the vein. The instantaneous death without any symptoms preceding it can only be explained by the fact that the air entered with force and in large quantity the right ventricle and arrested the heart's action by over-distension.

Internal Jugular. Ulrich's case.[2] The operation was performed for the removal of a tumor involving the left side of the neck. It was found that the tumor was attached to the deep vessels of the neck, and in severing its connection, the internal jugular vein was opened. No hemorrhage followed, the vessel remained open like an artery, and air entered immediately. The patient fainted;

[1] Medico Chirurg. Trans, vol. xvi. p. 29.

[2] Rust's Chirurgie, Berlin, 1836, vol. xvii. p. 565.

twitching of the muscles of the face, opisthotonos, a few slow respirations followed, and the patient was dead. The vein was found obliterated above the incision, and thickened, and more resistant than normal where the wound was inflicted. The reporter attributed the ingress of air to aspiration of the heart, and death to paralysis of this organ.

Remarks.—The pathological changes in the vein above the wound, interrupting entirely the column of blood from above as well as the thickening of the incised vein walls were potent factors which determined the entrance of air. As this operation was performed before anæsthetics were used, we may be almost certain that the patient was in a sitting or half-reclining position during the operation, thus favoring greatly venous return and ingress of air. The entrance of air in this instance is brought in connection with the suction power of the heart by the author in accordance with the prevalent doctrine of the French Commission.

Internal Jugular. Dupuytren's case.[1] The operation consisted in the removal of a tumor of a fibro-cellular character of considerable size from the neck of a female, 22 years of age. No serious obstacles presented themselves until the last deep attachment was severed with the knife when suddenly a prolonged hissing noise (*Soufflement prolongé*) was heard resembling the sound produced by the entrance of air into a vessel from which it had been exhausted. The patient immediately proclaimed "I am dying," and instantaneously dropped down on the floor, a lifeless corpse. As no other cause was found which could in any way account for the sudden death, the fatal issue was attributed to the entrance of air into the internal jugular vein. The following account of the post-mortem appearances fully warrant this supposition: "The right auricle was distended with air like a bladder, which rushed out when cut open without any admixture of blood. Fluid blood was found in the different vessels. Great quantities of air were found in all the vessels. There was no other unnatural appearance in any other part of the body."

[1] Medico-Chir. Trans., vol. xvi. p. 301.

Remarks.—The editor of the *Medical and Chirurgical Review* explained the entrance of air in this case as follows: "It proves that the heart acts as a sucking as well as forcing pump, otherwise air could never have passed from a cut vein in the neck down into the right chambers of the heart. It is highly probable that, in consequence of the morbid state of the parts, the mouth of the cut vein had remained patulous, and thus readily admitted the air." As no mention is made of the occurrence of hemorrhage, the vein was probably empty, a condition which might have been owing to the position of the patient during the operation or the pressure of the tumor. It is also reasonable to assume that, on account of the intimate connection of the tumor with the vessel, the former so altered the structure of the latter as to prevent closure of the wound, all of these causes combined resulting in aspiration of air during inspiration.

Facial Vein. Mott's case.[1] The operation consisted in extirpation of the parotid gland, the seat of a scirrhous tumor. The facial vein was opened, where it passes over the base of the lower jaw, in dissecting the integuments from the tumor in the early stage of the operation before a single artery was tied. At the instant this vessel was opened, the attention of all present was arrested by the gurgling noise of air passing into some small opening. The breathing of the patient at once became difficult and laborious, the heart's action violent and irregular, his features were distorted, and convulsions of the whole body soon followed to so great an extent as to make it impossible to keep him on the table. He lay upon the floor in this condition for nearly half an hour, as all supposed in *articulo mortis*. The convulsions ceased gradually, his mouth was distorted, and complete hemiplegia was found to have taken place; after an hour had passed he could speak, but the use of his arm and leg was only recovered completely after the lapse of a day.

Remarks.—Although not stated, it was undoubtedly true that in this instance the facial vein was enlarged, and its walls had lost their normal resiliency, thus favoring the ingress of air. This case is also of interest, as from the predominance of the

[1] Ibidem, p. 32.

cerebral symptoms it is apparent that some of the air must have passed through the pulmonary circulation and have gained access into the cerebral vessels from the left ventricle, giving rise to symptoms of cerebral embolism which disappeared as the air was absorbed.

Axillary Vein. Bransby Cooper's case.[1] The patient was a female, 19 years of age, who was the subject of a malignant tumor of the right humerus which required amputation at the shoulder-joint. The operation was done by making a double flap, the subclavian artery in the mean time being compressed against the first rib. There was no loss of blood. The subclavian artery was secured, compression being kept up, as there were small vessels which required ligation. As the operator was removing an enlarged gland from the axilla, he heard distinctly a peculiar gurgling noise, like air escaping with fluid from a narrow-necked bottle. At the same moment the patient fell into a state of collapse which threatened immediate death. The face was deadly pale, the pupils fixed and insensible to light, the pulse small and fluttering, at intervals regular, respiration hurried and feeble, and at irregular intervals attended with a sigh. The patient was placed in the recumbent position, the flaps closed, and stimulants applied; but an hour elapsed before she had sufficiently recovered to be removed from the operating room. Subsequently, when placed in bed, she maintained a constant motion of alternate flexion and extension of the right leg which continued for several days, at the same time she complained of pain, extending up the right side of the neck and head. The next day the pulse varied from 140–150 per minute, which remained the same for two days. She gradually rallied and recovered completely.

Remarks.—In this case the axillary vein was divided at a point where its walls are firmly fixed and its lumen kept patent by dense connective tissue which surrounds the vessel, a condition which predisposes to aspiration of air. Pulmonary air-emboli obstructed the passage of blood through the lungs, a circumstance which would serve to explain the rapid respiration and the accelerated action of the heart until the obstructing cause was removed.

[1] Med.-Chir. Trans., vol. xxvii. p. 41.

Axillary Vein. Courvoisier's case.[1] The operation was performed for the removal of a recurring cancer of the breast, and included the extirpation of infiltrated and ulcerated axillary glands. As the dissection reached the upper margin of the mass of axillary glands a lapping (schluerfendes) sound was suddenly heard; at the same time the patient, a robust woman 58 years of age, sank into a condition of collapse. The central portion of the vein was at once closed by digital compression, and artificial respiration with the administration of stimulants were successful in restoring her after the lapse of half an hour. Both ends of the vein were ligated, and the central ligature included the forceps, which were allowed to remain. The patient recovered.

Remarks.—In this instance the entrance of air was again determined by the anatomical location of the vein wound, to which may have been added cancerous infiltration of the paravascular tissues, which rendered the vein walls still more unyielding. The amount of air admitted must have been small, to judge from the evanescent nature of the symptoms which followed.

Anterior Thoracic Vein. Amussat's case.[2] The patient was a woman, 47 years of age, suffering from a scirrhous affection of the right mammary gland and the subjacent and surrounding tissues. The breast and adjacent tissues had been removed, and the operator was dissecting towards the opposite side, when suddenly, on making an incision into some suspicious granulations on the inner side of, and below the left clavicle, he and three other surgeons who were assisting him heard a sudden, distinct, interrupted sound, as of air passing into a cavity through a narrow opening. The patient exclaimed, "I am dying," and appeared to be suffocating. A repetition of the same sound convinced the operator that air had entered through a wounded vein, and he placed his finger on the spot from which the sound proceeded. The patient's condition became critical; a cold sweat covered her face, her eyes were turned upwards, and all around her thought her dying. The orifice of the wounded vein could be distinctly seen. The chest was compressed with a

[1] Correspondenz-blatt fuer Schweizer Aerzte, p. 205, 1880.

[2] Gazette des Hôpitaux, July 6, 1837.

view to force out the air from the vein, the wounded spot being compressed during the expansion of the chest. The patient soon began to show signs of improvement, when the operation was completed, and the vein with a portion of the tissue was tied. The patient recovered completely.

Remarks.—Although the particular vein wounded in this instance is not specified, it was undoubtedly a branch of the subclavian vein, the wound being in close proximity to the latter vessel. This case furnishes a good illustration of the fact, that veins of comparatively small calibre, when wounded near their proximal termination into a large vessel, may serve as points of entrance of air under the same circumstances as when the principal trunk is injured.

Superficial Cervical Vein. Trélat's case.[1] M. Trélat related, at a meeting of the Société de Chirurgie of Paris, an important case in which sudden death occurred in a patient from whom he was proceeding to remove a submaxillary tumor. The patient turned ghastly pale, and the heart's action ceased suddenly. Artificial respiration and electrization of the phrenic nerve induced some respirations and a slight return of color after fifteen minutes, but ineffectually. At the post-mortem examination a small vein opening into the external jugular was found to have been partially divided; in the jugular was a long clot interspersed with air-bubbles, and other bubbles of air were found in one of the mediastinal veins and the posterior cardiac vein, and a very notable quantity of air in the right chambers of the heart.

Remarks.—Several members of the society argued that death in this instance was due to the anæsthetic, and not to the entrance of air into the vein. Roux and Giraldès claimed that in several cases of death from chloroform they had found gases in the heart, in the vena cava, and even in the veins of the pelvis, but M. Depaul, in reply, properly and forcibly pointed out that the air in this case occupied only the veins going to the heart and the wounded vein.

[1] British Medical Journal, March 16, 1872.

Femoral Vein. The only well-authenticated case of aspiration of air into the femoral vein that I have been able to find is recorded in the *Medical and Surgical History of the British Army in Turkey and the Crimea*, vol. ii. p. 277, and refers to the sudden death from this cause of a soldier who had suffered amputation of the thigh. Three days and one-half after the operation he died suddenly without any obvious cause. At the necropsy, twelve hours after death, it was ascertained that the right cavities of the heart were distended with a mixture of blood and air, and the same condition was found in the two iliac veins and the inferior vena cava.

Remarks.—As the time which had intervened between the operation and the fatal accident was more three days, it is necessary to assume that the venous thrombus had been removed by suppuration, thus opening the vein for the admission of air, or that the supposed air found in the heart and vessels was not air but gas which had developed in the wounded parts, and had gained entrance into the venous circulation. This latter supposition is strengthened by the statement that the surfaces of the flaps were separated by gaseous products, and that the femoral vein was not closed but lay open on the surface of the stump.

Internal Saphenous Vein. Warren's case.[1] The operation was done for the removal of a tumor from the inner surface of the thigh. In the dissection the internal saphenous vein was wounded; the event was promptly announced by an audible and distinct sucking sound produced by the entering air. No alarming symptoms followed, as the further ingress of air was promptly prevented by closure of the vein.

Uterine Veins. That the entrance of air into the uterine veins might be a cause of danger after parturition was suggested by Legallois in 1829. Dr. John Rose Cormack read a paper on this subject before the Westminster Medical Society in 1850 when he gave the details of three cases that had occurred in his neighborhood. Many authorities doubt the possibility of admis-

[1] Gazette Médicale, No. 52.

sion of air into the uterine veins after labor. Julius M. Kolb[1] alludes to this subject as follows: "I have not seen a case which convinced me that air had passed into the open veins of a recently delivered uterus, and I cannot conceive the mechanical possibility of such an occurrence." Bessems, Lionet, Lever, Wintrich, Berry, and Simpson assert that they have met with such instances, and a sufficient number of well-authenticated cases have been placed on record which leave no further doubt as to the possibility of sudden death in puerperal women from entrance of air into the uterine sinuses. In a recent number of the *Wiener Medicinische Zeitschrift*, Braun gives three fatal cases from the introduction of air into the uterine veins; in two of three the uterine douche was used, in one to produce abortion, in the other after delivery of twins, and the patients died in a short time, one indeed within twenty minutes. Post-mortem examination showed air in the uterine veins, in the ascending vena cava, and in the veins of the heart. The third case was that of a woman who had been delivered lying upon her left side, and was then turned upon her back; massage was made over the uterus, she gasped and died in a few minutes. Braun suggests that in the change of position a volume of air entered the uterus, and the manipulation, instead of driving it out, forced it into the uterine veins. Bischoff refers to two cases that came under his observation.[2] Dr. Draper[3] has reported two cases where instant death occurred from efforts to cause criminal abortion. The post-mortem examination proved in each case the presence of air in the veins.

Mr. George May[4] reports three cases which occurred in his vicinity. The patients died respectively immediately, six hours, and eight days after delivery, and in all of them post-mortem examinations showed the presence of air in large quantities in the inferior vena cava and the right side of the heart. An inte-

[1] The Pathological Anatomy of the Female Sexual Organs, 1868, American Translation.

[2] Corresp. bl. f. Schweizer Ärzte, p. 206, 1880.

[3] Boston Med. and Surg. Journal, January, 1883.

[4] British Med. Journal, June 6, 1857.

resting account of this accident is given by Dr. George Cordwent, and relates to a case that came under his observation.[1]

His patient was 28 years of age. During the delivery, at full term, her expulsive pains became urgent, and at her request she was permitted, in the absence of her medical attendant, to remain standing; after a few severe pains the child was expelled, and after falling on the floor dragged with it the whole placenta. Almost immediately afterward a kind of gurgling sound was heard by the attendants, but whether it arose from rumbling in the bowels they could not say. The patient remained about one minute standing as before and holding on to the bed-post; she then cried out: "I can't see! I feel faint! lay me on the bed," and expired almost instantly. At the necropsy, 24 hours after death, it was shown that the uterus externally presented the normal appearances of a recently delivered organ, except that a portion of the wall of its fundus to about the extent of a five-shilling piece was slightly more puffy than the other portions, and, on cutting into it, air-bubbles escaped. There had been no laceration of the placental surface; the uterine cavity contained only one small clot; its lining membrane was healthy. The coronary vein of the stomach was distended; the right side of the heart was slightly gorged, and when the auricle was punctured air-bubbles escaped with the blood which it contained.

Davidson[2] reports the case of a Hindoo woman who was admitted into the Kaira Gaol Hospital and safely delivered of a female child. The labor was in every respect normal. The placenta came away at the usual time, and there was no post-partum hemorrhage. About three-quarters of an hour afterward the woman died without any apparent cause. There had been no hemorrhage or convulsions. The patient had been taking some nourishment when she suddenly fell back and expired. At the post-mortem examination two hours after death the uterus was found empty, with large and somewhat distended veins; the right side of the heart contained a quantity of air mixed and churned up with blood, which escaped in bubbles,

[1] St. George's Hosp. Reports, vol. vi.
[2] The Lancet, vol. i. p. 999, 1883.

the lungs were congested; all the other organs were normal. The most interesting and convincing case is related by Olshausen. It is the most convincing on account of the painstaking and accurate post-mortem examination which was made to determine the cause of death.[1]

A robust secundipara, aged 29, was delivered at full term. The uterus was unusually distended; no albumen in urine. The labor was lingering and the uterine douche was used. The water was of 30° R. and was forced into the vagina gently by a pump. A third injection was made by a midwife. After eight minutes' use the patient began to complain of oppression. The tube was withdrawn. The patient rose in bed, immediately fell back senseless, and died in less than a minute under convulsive respiratory movements and distortion of the face. Eight minutes later bleeding by the median vein was tried, but only a few drops flowed. On touching the body distinct and widespread crepitation was felt.

Autopsy eight hours after death.—A large quantity of dark fluid blood escaped from the sinuses of the dura mater. The cerebral membranes very hyperæmic; brain normal, lungs somewhat congested, heart lying transversely, apex in fourth intercostal space. Left ventricle in firm contraction, right quite soft, something like an intestine with thick walls; the coronary vessels contained a quantity of air bubbles. Left heart contained only a small quantity of blood; the right held little, but it was frothy. The distended uterus crepitated everywhere on pressure under the hand. A number of subperitoneal vessels of medium size were plainly filled with air. The right broad ligament was strongly distended with air bubbles, and this emphysema of cellular tissue extended from the broad ligament through the retro-peritoneal space to the inner side of the right kidney, and even below the liver to the inferior vena cava. *The inferior vena cava was enormously distended*—it was at least an inch in diameter—containing mostly air. The uterus was divided in the median line; a placenta was attached to the anterior wall; a small flap was detached from the uterus, a second placenta was attached behind and to the right; a larger portion of this had been separated, so that there was a sort of a pouch between it and

[1] Monatsschrift f. Geburtskunde, Jan. 1865. American Journ. Med. Sciences, July, 1865.

the anterior wall. The two ova were uninjured. The air had gained access into the veins at the placental site. It was concluded that the tube had been passed into the uterine cavity and that air had been thrown in with the water by the pump.

It would be difficult to conceive in what manner air could be drawn into the uterine veins by the aspiratory movements of the chest or heart, as is the case in the veins about the apex of the chest. Another explanation must be sought for, and this will be found in the change of structure, and the relations of the uterine veins. The veins during pregnancy keep pace with the enormous physiological hyperplasia of the uterine tissues, and are gradually converted into large sinuses, more especially the vessels at the placental site; they are simply excavations or channels in the contractile muscular walls of the uterus, their size being subject to the state of the uterine walls, whether at rest, relaxation, or contraction. When the placenta is detached, some of these sinuses are laid open, and in a normal condition their calibre is obliterated by the contractions of the uterus and the formation of thrombi. If, from any cause, air should reach the uterine cavity, it may be aspirated into the uterine sinuses by relaxation of the uterine contractions, and, having gained access into them, it is readily forced into the circulation by subsequent contractions; the uterine walls acting the part of a suction and forcing-pump. During forcible uterine contractions the veins are nearly emptied of their contents, and, as the organ relaxes, the walls of the veins are distended and a vacuum is formed, which is filled with blood or air. Should the relaxation be slow, the empty spaces are readily filled with blood or serum in the absence of air, but if the uterus relaxes quickly the suction power is proportionately greater, and, in the event air has reached the uterine cavity, it is aspirated into the open veins, and, by reaching the right side of the heart through the vena cava, it gives rise to the same train of symptoms as when it is admitted into a vein during a surgical operation in the regions of the neck.

Pulmonary Vein. Dumin's case.[1] This is the only case on record where it is claimed that death was produced by the entrance of air from a pulmonary tubercular cavity through the pulmonary vein into the left side of the heart. The patient was a young man suffering from pulmonary tuberculosis in the last stage. Physical diagnosis revealed a large cavity in the apex of the right lung. After the patient had been in the hospital for three weeks the general conditions remained about the same, while the local destructive process had been progressing. One day, after eating his dinner, he arose from his bed, fell down, and expired almost instantly without uttering a word or sound. At the post-mortem examination, twenty-four hours after death, it was found that the apex of the lung contained a cavity of considerable size, besides extensive crude infiltrations. The left lung contained numerous nodules and three small cavities. The third cavity in the substance and near the base of the lung contained a small amount of blood intimately mixed with air-bubbles. The heart was slightly dilated. The left ventricle was filled with blood mixed with innumerable small air-bubbles. The right cavity also contained air, but in much lesser quantity. All the larger arteries contained air mixed with blood; air-bubbles were also found in the venæ cavæ and the pulmonary artery. The arteries and veins in the brain and meninges were found distended almost exclusively with air. No signs of advanced putrefaction could be found, and none of the parenchymatous organs contained gases. The reporter explained the sudden death by the entrance of air from the small cavity in the left lung, which contained spumous blood, the air having found its way into an open branch of the pulmonary vein, and from thence into the left side of the heart. The air, which was found in the right side of the heart and veins, according to his view, had passed through the systemic capillaries. As the direct cause of death, anæmia of the brain is mentioned.

Remarks.—It seems to me that several reasons might be mentioned which would throw doubt on the correctness of the assertion that, in this case, the immediate cause of death was owing to entrance of air into the pulmonary vein. 1. The time which had elapsed from the commencement of the attack until

[1] Berliner Klin. Wochenschrift, January 30, 1882.

death took place was not sufficient to produce such an extensive distribution of air, unless it could be proved that the heart's action continued after respiration had ceased. 2. The existence of an open vessel in any of the cavities was not proven at the examination after death. 3. The body appears to have been affected by a certain amount of putrefaction, which may have been sufficient, in degree, to give rise to the evolution of gases, and the putrefactive changes may have been limited to, or were at least farthest advanced in, the blood, which would explain the absence of gas in any other part of the body, except within the bloodvessels. 4. Snycope is a frequent cause of sudden death in greatly debilitated patients when the heart is called upon to perform an increased amount of labor, as when the patient suddenly assumes the erect position.

Superior Longitudinal Sinus. Volkmann's case.[1] The only fatal case of admission of air into the sinuses of the dura mater is reported by Genzmer. The patient was a female, 63 years of age, who was affected with a perforating sarcoma of the dura mater. The tumor was noticed about two years before the operation, and was located in the region of the posterior extremity of the sagittal suture, and for a long time gave rise to no inconvenience. For the last six months it caused intense headache. On one occasion, a physician believing that it was an atheroma, attempted its removal, but as the first incision gave rise to copious hemorrhage, he desisted from any further attempts, and the wound healed kindly. When the patient was admitted under Volkmann's care into the Clinic at Halle, the tumor presented a lobulated appearance, being composed of three parts, each about the size of a plum, and was located over the posterior extremity of the sagittal suture. On touch, the tumor was soft and elastic, and imparted to the finger distinct pulsations. Gradual compression reduced its size one-half; when the pressure was discontinued it resumed its further dimensions. On auscultation, a blowing sound was heard synchronous with the radial pulse. By pressing the end of the index finger deeply between the lobes of the tumor, a bony defect in the skull was readily detected. The conclusion was reached, that the tumor had sprung from the dura

[1] Verhandlungen d. Deutschen Gesellschaft f. Chirurgie, vol. vi. p. 32.

mater, and had perforated the skull by the prolonged pressure, causing interstitial absorption of the cranial vault. During the patient's stay in the hospital the tumor increased very rapidly in size. As no brain symptoms were present, it was assumed that the substance of the brain was intact. In view of the speedy fatal issue, which of necessity would take place without operative interference, Volkmann decided to remove the tumor. The operation was done April 2, 1875. Under strict antiseptic precautions the tumor was exposed by a crucial incision, and the flaps reflected with the periosteum to the margins of the opening in the skull. The aperture in the bone measured $5\frac{1}{2}$ by $4\frac{1}{2}$ cm. in diameter. With a Luer's cutting forceps the opening was enlarged to 7 by 8 cm. The tumor, when exposed, was nearly as large as a fist, and firmly adherent to the dura mater. The dura mater was carefully divided around the margins of the tumor, which had now been liberated from all its attachments except the falx cerebri. It was now drawn forward through the opening in the skull, and the falx cerebri divided with scissors from before backwards. This step of the operation was attended by alarming hemorrhage. As the blood was being sponged away to expose momentarily the field of operation, a peculiar and characteristic lapping sound was heard, which indicated to all present that air had entered the longitudinal sinus. At the same time the assistant, who was giving the chloroform, remarked, "She is dying." The wound was immediately compressed with a large carbolized sponge. The patient was in collapse, her breathing was interrupted and stertorous. After a short pause, it was determined to complete the operation, but as soon as the tumor was again drawn forward, and its attachment at the junction of the longitudinal with the transverse sinuses was divided, air again entered, accompanied by the same characteristic sound. The tumor was separated rapidly from its remaining attachments, and a Lister dressing was applied in such a manner as to make at the same time a requisite amount of compression for the double purpose of arresting hemorrhage and preventing further ingress of air. At this time the patient was pulseless, pupils dilated, extremities cold and blue. Auto-transfusion, by constricting the arms and legs with elastic bandages, had the effect of momentarily stimulating the heart, but respiration became more irregular and interrupted, and after a few more brief moments the patient died. At the post-mortem examination, which was held on the following day, the right side of the

heart was opened under water, air-bubbles escaped, showing conclusively that air had made its entrance through the longitudinal sinus. The left side of the heart contained no air. Air was also found in the pulmonary artery and the subpleural vessels. The left side of the brain had suffered more from compression by the tumor than the right. The defect in the dura mater corresponded to the opening in the skull. An additional source of hemorrhage was detected at the posterior margin of the defect in the cranium, where the opening of a vein in the substance of the bone, 5 mm. in diameter, could be seen. Under the microscope the tumor showed small spindle-shaped cells, with a very vascular intercellular substance.

Remarks.—In this case all circumstances favored the entrance of air into the wounded sinus. The sudden and severe loss of blood from such a large reservoir as the longitudinal sinus rendered the vessel empty, thus creating the most essential element in the causation of air aspiration. The position of the patient during the operation undoubtedly was such that the force of gravitation assisted materially in the formation of a vacuum. The walls of the sinus being rigid and attached to the surrounding structures prevented collapse of the vessel, and held the wound patulous. That death was owing to the introduction of air is sufficiently proven by the symptoms during life and the evidences derived from the post-mortem examination.

Veins of Diploë. Franck[1] asserts that he has repeatedly seen aspiration of air into the veins of the diploë after trephining. He claims that the air reaches the heart through the medium of the vertebral veins, which, from their protected position, are more favorably located for this purpose. By experiments he proved that ligation of the jugular veins does not prevent the aspiration of air through the veins of the diploë, while, on the other hand, this accident cannot happen when the vertebral veins are compressed. As the veins of the diploë in some instances are unusually large, and their walls firmly attached to

[1] Sur la transmission de l'aspiration thoracique jusqu' aux canaux veineux des os du crâne, etc. Gazette Méd., No. 25, 1881.

the unyielding bone tissue they constitute channels which cannot contract in the event they are injured, consequently we should *a priori* expect that aspiration of air will take place under the same circumstances as in the case of the sinuses of the dura mater, and in all extensive injuries of the cranial bones the same caution should be exercised to guard against this accident. In troublesome hemorrhage from venous sinuses in bone, the bleeding is promptly and safely arrested by implantation of an aseptic sponge, which can be left *in situ*, as it will be removed by the granulation tissue during cicatrization. In such instances the sponge is peculiarly well adapted, as the lumen of the vessel is surrounded by unyielding bony walls, which will support any amount of pressure on part of the aseptic tampon.

XI. *Experiments on Venous Air-Embolism.*

The injection of air was always made into the jugular vein. The neck was shaved, and the surface disinfected with a five per cent. solution of carbolic acid. Ether was always used as an anæsthetic, the animal being kept fully under its influence until everything was in readiness to thrown in the air, when the inhalation was suspended, and the animal was allowed to come out from under its influence, for the purpose of studying the effects of the air on the heart and respiration, independently of the effects of the anæsthetic. The vessel was freely exposed, usually in the lower part of the neck, by a parallel incision. After isolating it to the extent of from two to four inches, the influence of the respiratory movements of the chest on the venous circulation was carefully studied. Then a hæmostatic forceps was applied to the distal portion of the vein. Below the point of compression the blood was forced out of the vessel between two fingers, and its return prevented by applying another pair of forceps to the proximal end of the exposed vein. We had thus a bloodless portion of vein between the forceps, presenting a ribbon-like band. This was partially divided in an oblique direction for the purpose of facilitating the introduction of a canula. The canula was securely fastened in the vein by a

ligature, when the proximal pair of forceps were removed, and, by compressing the bulb, the air was injected with force, so as to imitate as nearly as possible the conditions present during the accidental introduction of air. The canula was connected with a rubber-bulb, of known capacity, by means of a rubber-tube. After the completion of the experiment (if the animal survived) the vessel was divided completely, and both ends ligatured with catgut, and the wound closed with a continued catgut suture. The weight of the animal and amount of air injected were estimated accurately in most instances.

Experiment No. 1.—Sheep, weighing 120 pounds. Left jugular vein. The vessel was opened in its lower third, but no air entered. A rubber-tube was introduced for a distance of two inches with a view of facilitating the spontaneous ingress of air, but this accident failed to occur. Air was injected, at intervals of eight minutes, in quantities of 30 c. cm each, until the enormous amount of 480 c. cm. had been introduced. After the first injection nothing was observed that indicated the presence of air in the veins or the heart. After the second dose a slight splashing sound could be heard over the cardiac region, which became louder and more distinct as the amount of air in the right side of the heart increased. The first serious symptoms observed were a tumultuous action of the heart and difficulty in breathing, which became aggravated by every succeeding injection. Towards the end of the experiment, which lasted nearly two hours, the animal was attacked, at short intervals, by general convulsive movements. After the suspension of respiration the heart's action became very slow and feeble, and at times irregular. The immediate cause of death was plainly due to asphyxia, as manifested by the great dyspnœa and the cyanotic hue of all visible mucous surfaces. On examination after death, a few air-bubbles and only a small amount of dark blood were found in the left ventricle. The right ventricle was arrested in the diastole and contained a large quantity of very dark, almost black, spumous blood. Air-bubbles were found in a number of distant arteries of small size.

Experiment No. 2.—Adult, large cat. In this instance the canula was introduced and tied in the left jugular vein. The heart was

exposed before the injection was made with a view of observing directly the effects produced by sudden inflation of the right cavities of the heart. Before the air was introduced, the heart contracted regularly—artificial respirations being made for the purpose of preventing death by asphyxia. As soon as the right side of the heart was distended by the air, the left auricle and both ventricles ceased to contract, while the right auricle continued to pulsate. The pulsations were feeble and irregular. The coronary veins became filled with air-bubbles, presenting the appearance of a rosary. On opening the superior vena cava, air and frothy blood escaped, the right side of the heart collapsed and all chambers of the heart commenced to contract regularly and with considerable force. The pulsations continued for 15–20 minutes, becoming more feeble and irregular and intermittent towards the last. After death, air was found in both venæ cavæ and the iliac veins. The left ventricle was completely empty. In this case, owing to the small size of the heart and the large amount of air introduced, the contractions of the right ventricle were arrested in the diastole, while respiration continued. Death took place suddenly from mechanical over-distension of the heart.

Experiment No. 3.—Dog, weight 65 pounds. Injected 30 c. cm. of air into left jugular vein. Churning sounds over cardiac region loud and distinct. Heart's action became very tumultuous and intermittent. Respirations superficial and rapid. The animal was bled from the distal end of the jugular vein to the amount of four ounces, whereupon the heart's action became regular and the respirations diminished in frequency. The vein was divided completely and both ends were tied with fine catgut ligatures, the wound being closed in the usual manner. For a number of days the dog appeared quite unwell, showed no disposition to eat and acted very stupidly, being inclined to sleep most of the time. Subsequently he recovered completely. In this case the intravascular pressure was promptly relieved by free bleeding which enabled the heart to force the air through the pulmonary into the general circulation. The stupid condition of the animal was undoubtedly owing to embolism of the cerebral vessels, which disappeared after the disappearance of the air-emboli by absorption.

Experiment No. 4.—Adult, medium-sized cat. Injected 15 c. cm. of air into left jugular vein. Heart's action arrested at once.

Respirations which were irregular ceased a few moments later. The chest was opened at once. The right side of the heart was found enormously dilated and almost motionless. Coronary veins filled with air. The right ventricle was punctured with the needle of an aspirator and its contents withdrawn, when the pulsations were re-established. The ventricle was again inflated through the needle of the aspirator. Five minutes after this injection the pulsations numbered about 250 per minute. Five minutes later the left auricle ceased to contract, the movements of the right being irregular and about 80 to the minute. After the lapse of another five minutes the pulsations of the ventricles were only 17 a minute and a little later all movements ceased. This experiment demonstrates that the arrest of the heart's action was due to mechanical over-distension, as aspiration of the right ventricle was followed by regular and strong contractions in all cavities of the heart. The contractions were not the result of the mere mechanical irritation of the heart by the puncture, as other and equally severe irritants were previously applied without producing any effect.

Experiment No. 5.—Dog, weight 35 pounds. Before operation, respirations 40, pulse 140. Injected 20 c. cm. of air into right jugular vein. Convulsions followed which lasted for about two minutes. Respiration rapid and stertorous. Pulse 300. After five minutes the animal made repeated attempts to get up and walk, but invariably fell down on account of imperfect control over the movements, or paralysis of the posterior extremities. Half an hour later the animal was able to walk, but appeared very feeble. Pulse 120, respirations somewhat accelerated. Recovery was complete. In this case the equilibrium of the circulation was soon restored and the air in the right side of the heart passed through the pulmonary into the general circulation in a very short time, as was evident from the presence of symptoms indicative of embolism of some of the vessels in the cerebro-spinal centres.

Experiment No. 6.—Dog, weight 75 pounds. Injected 60 c. cm. of air into the right jugular vein. Churning sounds loud and distinct; heart's action labored. Respirations exceedingly rapid, later stertorous. The animal recovered rapidly from the immediate effects of the air-embolism, and was soon as well as before the operation.

Remarks.—These experiments tend to prove the following statements: 1. A small amount of air in the right side of the heart in a healthy animal gives rise only to temporary symptoms referable to the heart's action and the pulmonary circulation. 2. When air has been introduced into the right side of the heart in such quantities as not to arrest the contractions of the heart at once, it is forced through the pulmonary capillaries into the left side of the heart by the contractions of the right ventricle. 3. The danger attending the insufflation of air into veins is proportionate to the amount of air introduced, as well as to the capacity of the right ventricle to resist intra-cardiac pressure. 4. When a fatal dose of air has been introduced into the venous circulation, death takes place almost instantaneously from arrest of the heart's action, or later from suffocation. 5. Spontaneous ingress of air into a wounded, healthy jugular vein never occurred in these experiments and must be considered almost a physical impossibility, as the resilient walls of the wounded vein collapse readily when exposed to atmospheric pressure.

Dogs weighing about 30 pounds would usually recover in a short time after an injection of 30 c. cm. of air, while double that amount constituted generally a fatal dose. Sheep required a proportionally larger dose to produce a fatal result. These experiments also tend to prove that animals of the same kind and species do not manifest the same degree of tolerance to the presence of air in veins. Young animals succumb more readily to its effects; in the adult animal the degree of tolerance depends on the development and contractile power of the right ventricle. In all cases where life was prolonged for a considerable length of time after the injection of air was made, and the ventricular contractions were not much impaired, bubbles of air were found in the left side of the heart, showing conclusively that the air must have passed from the right ventricle through the pulmonary capillaries. In experiments Nos. 3 and 5 the well-marked cerebral disturbances were undoubtedly produced by secondary air-embolism of the vessels of the brain and spinal cord, since these disturbances disappeared after the removal of the emboli by absorption. When the same amount of air was administered in

one dose, it always proved more dangerous than when injected in divided doses, which simply means, that during the interval between the injections sufficient time had elapsed for the right ventricle to force at least a portion of air through the pulmonary capillaries into the general circulation; thus preventing for a time at least a fatal degree of intra-cardiac pressure. The greater the development of the right ventricle, the greater the tolerance of the animal to the presence of air in veins. This statement is well exemplified by the fact that in horses a larger amount of air is required to produce death, while in dogs a proportionately much smaller dose will result in death. This is because in the former animals the right ventricle, from its proportionately greater strength, is more competent to perform an additional task. If death followed immediately after the injection, the dose was usually a large one, the heart's action having been suspended before respiration ceased, and on post-mortem examination the right ventricle and auricle were always found over-distended and tympanitic, containing a moderate amount of blood not intimately mixed with a large amount of air, while the left side of the heart was nearly or completely empty. If death occurred some time after the injection, great dyspnœa was observed, lividity of the mucous membranes, tumultuous action of the heart, and respiration ceased prior to cessation of the action of the heart, showing conclusively that death took place from carbonic acid intoxication. In these cases the necropsy revealed a lesser degree of distension of the right chambers of the heart, which invariably contained spumous blood or blood intimately mixed with air. The pulmonary artery and its branches were filled with air and spumous blood, and bubbles of air and a small quantity of blood were present in the left ventricle. In opening the jugular vein I have always noticed that the vessel would promptly collapse as soon as hemorrhage ceased, being transformed into a pale, round cord, in which the wound could be found only with great difficulty. Air was never seen to enter, even if the wound and a portion of the vein were kept patent with forceps or a rubber-tube. I frequently arrested the column of blood in the vein by applying a compressing forceps

above, and opening the vessel below, thus creating a favorable condition for the spontaneous entrance of air, but the result remained the same. It is therefore only reasonable to assume, that air will enter spontaneously only in case a vein near the heart is wounded, the walls of which are prevented from collapsing by fixations of its tunics to adjacent, unyielding structures, or in case the vein walls themselves have lost their resiliency by previous pathological changes.

XII. *Experiments on Arterial Air-Embolism.*

Experiment No. 1.—Medium sized dog. The left carotid artery was exposed and isolated, and two hæmostatic forceps applied about two inches apart. The artery was divided between them, and the proximal end secured by a ligature. Into the distal end the canula of the injecting bulb was introduced and fastened with a ligature. When the animal had fully recovered from the anæsthetic, the forceps were removed, and 80 c. cm. of air were injected at once and with considerable force; the canula was then removed and the artery ligated. The animal collapsed almost instantaneously, and respiration was suspended for nearly two minutes. The animal appeared motionless and in a condition of profound stupor. Heart's action tumultuous and very rapid. Two minutes after the injection churning sounds were audible over the cardiac region. When respiration was re established the movements of the chest were slow and irregular, gradually becoming slower and slower until, after about fifty attempts, they ceased entirely. The limbs were rigid, and the trunk in a position of opisthotonos. The heart continued to contract for about two minutes after respiration had ceased. All vessels leading to and from the heart were carefully ligated, and the organ removed and examined under water. The coronary veins contained bubbles of air. All cavities of the heart contained air, the largest amount being found in the left auricle. Coagula were present in all of the chambers, except in the left auricle. The circle of Willis was distended with air, as were many of the smaller vessels of the brain and its membranes. The cerebral vessels were gorged with dark blood. The air injected into the carotid artery had evidently passed directly through the cerebral capillaries into the venous circulation, giving rise to venous and general air-embolism. In the

beginning respiration was immediately suspended from temporary suspension of innervation, but was re-established as soon as the amount of air in the cerebral vessels had lessened sufficiently to allow a better blood supply. It, however, ceased definitely under symptoms expressive of extensive embolism of the pulmonary artery.

Experiment No. 2.—Sheep, weight 90 pounds. Left carotid artery prepared as in previous experiment, only that the air was injected in a central direction, towards the heart. After the animal had recovered almost completely from the effects of the anæsthetic, 150 c. cm. of air were thrown in, and the proximal end of the artery was tied. The animal was at once thrown into a tetanic state, with rigid limbs and retracted neck. All reflex movements and sensations were completely suspended. The heart's action was irregular, tumultuous, and over the cardiac region distinct churning sounds were heard. Respirations exceedingly rapid. The mucous membranes accessible to sight presented a strikingly pale and anæmic appearance. Death occurred in fifteen minutes, respiration ceasing first. Examination immediately after death showed the right ventricle greatly distended and tympanitic on percussion. It contained spumous blood, air, and a few small coagula. Left ventricle contained only a minute quantity of spumous blood and fine blood clots adherent to the endocardium. Air-emboli were found in almost all vessels throughout the body. Coronary arteries distended with air. Jugular veins contained more air than blood. Basilar artery and superior longitudinal sinus contained a large amount of air. The tongue and other distant organs extremely anæmic. Although this large quantity of air was thrown directly into one of the large arteries, it was forced into the smallest vessels and through the capillaries into the veins and the right side of the heart by the powerful contractions of the left ventricle, giving origin to a combination of symptoms arising from arterial and venous embolism. Death was finally produced in a similar manner as if air had been injected directly into the veins. Some of the air had passed all the capillaries and was found in the left side of the heart.

Experiment No. 3.—Medium sized adult cat. With a view to ascertain how soon air would pass through the capillary vessels after injecting it into the carotid artery, the jugular vein on the opposite side was opened before the injection of air was made.

About 15 c. cm. of air were thrown into the carotid artery in a peripheral direction. The animal was immediately seized with convulsions and died in less than a minute. Respiration ceased first. Air was seen to escape from the wound in the jugular vein, and was also found in nearly all of the vessels throughout the body. On opening the chest the right side of the heart was found distended to its utmost, and tympanitic on percussion. The contractions of the right auricle were twice as rapid as the ventricular contractions. The movements of the left auricle were the same in frequency as the ventricular pulsations. The left auricle ceased to contract seven minutes after death. The right ventricle was punctured with the needle of an aspirator, and its contents removed, whereupon the contractions became much stronger.

8 minutes after death ventricles contracted 44 times a minute.

10	"	"	"	"	"	30	"	"
12	"	"	"	"	"	16	"	"
18	"	"	"	"	"	16	"	"
29	"	"	"	"	"	6	"	"

Then all contractions ceased and could not be restored by any kind of irritation.

Right auricle ceased beating ten minutes after death, but commenced to contract again nineteen minutes later, contracting 24 times a minute, the movements becoming more rapid and at times irregular for forty-eight minutes after death.

Experiment No. 4.—Dog, weight 35 pounds. Into the left carotid artery 60 c. cm. of air were injected in a proximal direction. The animal was seized at once with convulsions of a tetanic character with the limbs extended and rigid. Involuntary discharges from bowels and bladder. Sensation, motion, and reflex actions suspended. Pupils contracted. Respirations very rapid. Heart's action slow and labored. The left jugular vein was opened and four ounces of blood were abstracted with the result of greatly improving the heart's action. Both vessels were ligated and the wound closed. The animal was in profound stupor, resembling complete anæsthesia, until death occurred two and a half hours after the operation. Half an hour after the injection the pulse was 74, respirations 44 in a minute. On opening the abdomen the large veins were found very much dilated and contained air. Large air bub-

bles were also found in the pulmonary artery, internal mammary, and coronary arteries. Small thrombi were found in many of the ultimate branches of the pulmonary artery. The right side of the heart was distended with spumous blood and air. The left ventricle was almost completely empty, the endocardial lining presenting many patches of subserous ecchymoses. Nearly all vessels of the brain and the longitudinal sinus contained air. Cerebral and meningeal vessels engorged with blood. The animal died with marked symptoms of asphyxia. The tetanic rigidity of the extremities and the muscles of the trunk, as well as the remaining prominent cerebral symptoms, were produced by the intense cerebral congestion, the result of obstruction of some of the smaller vessels by air-emboli.

Experiment No. 5.—Adult dog, weight 20 pounds. Into the peripheral end of the right carotid artery 20 c. cm. of air were injected after the animal had completely recovered from the anæsthetic. The dog collapsed almost immediately and fell to the floor, perfectly unconscious, with extended and rigid limbs. Breathing exceedingly rapid, but gradually growing slower as consciousness returned. When the animal attempted to walk it staggered, and frequently fell, having apparently lost the power of co-ordination in the posterior extremities. The pupils, at first dilated, later became very much contracted. After about two hours, the animal walked without difficulty, but respiration, as well as the heart's action, continued very rapid. The next day complete recovery had taken place. Aside from the cerebral and spinal symptoms, the arterial embolism in this case resulted in no serious consequences; the most threatening symptoms were referable to venous embolism, showing that most of the air had passed through the capillaries into the venous circulation and right side of the heart.

Experiment No. 6.—Adult dog, weight 22 pounds. Right carotid artery exposed and 45 c. cm. of air injected towards the heart. Involuntary discharges from bladder and rectum. Pupils dilated, later contracted. Animal completely unconscious, anterior limbs extended and rigid, slight opisthotonos, breathing mostly abdominal. Churning sounds over heart heard a few seconds after injection, disappeared after eight minutes. Respiration ceased five minutes after the air was injected, but was again restored by artificial respi-

ration and faradization of vagus and diaphragm. Twenty-three minutes after the injection, pulse 128, respirations 24; seventeen minutes later, pulse 105, respirations 26; fifty-two minutes after injection, pulse 120, respirations 20; ninety minutes after injection, pulse 180, respirations 20. At this time the animal made several unsuccessful attempts to rise, but relapsed into a comatose state, which continued until death fifteen hours after the operation. At the post-mortem examination the arteries contained a good deal of dark, almost venous blood interspersed with bubbles of air. Air was found in the uterine, ovarian, iliac and mesenteric arteries and aorta; also in both venæ cavæ, pulmonary artery and veins. Some of the smallest branches of the pulmonary artery were obstructed by thrombi. Right ventricle distended with very dark, almost black, frothy blood. Left ventricle firmly contracted and almost empty. Left auricle contained dark, spumous blood. Coronary arteries distended with air; internal mammary arteries and veins also contained it. Air was found in all the cerebral vessels and sinuses which were distended with dark fluid blood. Vessels of spinal cord were intensely congested and contained numerous air-bubbles. The mucous membrane of the stomach presented eleven points of extravasation, the largest being circular and one inch in diameter. This case presented the most diffuse form of embolism, the emboli being found equally numerous and diffuse in the arterial and venous systems. The multiplicity and diffusion can be satisfactorily accounted for when we take into consideration the large amount of air which was injected, and the length of time which had elapsed from the time of insufflation until death occurred. The post-mortem appearances plainly indicated that death was produced by slow asphyxia. The primary unconsciousness and stupor were induced by the acute cerebral ischæmia; then a short period of consciousness returned as soon as the collateral circulation in the brain had become partially established, when again the animal became comatose from carbonic acid intoxication. The abstraction of blood relieved the threatening symptoms attending the arterial embolism, but failed in preventing death from asphyxia by secondary venous embolism.

Experiment No. 7.—Dog, weight 26 pounds. Injected 15 c. cm. of air into the right carotid artery in a peripheral direction. The animal had a slight convulsion and remained unconscious for about twenty minutes, when it rallied and was able to walk about the room

with a slightly staggering gait. No complications followed the operation. In this instance the cerebral symptoms followed the insufflation instantly, but owing to the small amount of air injected, they were only of short duration, the air—passing the capillaries in a short time—entered the venous circulation where it produced temporary disturbances in the pulmonary circulation.

Remarks.—Injection of air into arteries produces well-marked and characteristic symptoms which point directly to a disturbance in the circulation of the brain and spinal cord. The most prominent symptoms are: Convulsions, coma, tetanic rigidity of the limbs and extensor muscles of the back. The coma resembles complete and profound anæsthesia. These symptoms follow the operation more quickly if the injection is made in a peripheral direction toward the brain. If the animal does not succumb to the primary effect of the air upon the brain and medulla oblongata, a series of symptoms succeed which announce the arrival of air in the veins and right side of the heart. All of the experiments show that it only requires a few seconds for the powerful contractions of the left ventricle to force at least a large portion of the air through the capillaries into the veins. The primary effect of embolism of the vessels of the brain is to produce acute cerebral ischæmia, the intensity of which depends upon the number and size of the vessels which have become obstructed by the air-emboli. This anæmia soon gives rise to intense engorgement of the collateral vessels, and the vessels behind the point of obstruction, an engorgement to such an extent, that it often leads to rupture of capillary vessels and hemorrhage into the paravascular tissues. Unless the amount of air injected is sufficient to prove rapidly fatal by causing suspension of functions of the cerebro-spinal centres, the animal dies of asphyxia from embolism of the pulmonary artery, the same as though the air had been injected directly into the veins. The left ventricle, from its greater thickness and consequently more powerful contractions (as compared with the right ventricle), is better adapted to overcome the increased resistance, and hence the air is rapidly forced through the systemic capillary circulation into the veins and right side of

the heart. On this account an equal amount of air injected into arteries is not so dangerous as when introduced directly into veins, as a certain percentage of it never reaches the veins and right side of the heart. Arterial air-embolism is attended by an additional source of danger, which consists in the greater tendency to coagulation of blood in the heart and vessels, as was noted in several experiments.

XIII. *Direct Intra-Cardiac Insufflation of Air.*

These experiments were made with a view of demonstrating by ocular inspection, that sudden over-distension of the cardiac muscles will arrest their contractility and will thus produce paralysis of the heart in the diastole.

Experiment No. 1.—Medium sized cat. After the animal was fully under the influence of ether, respirations and heart's action being normal, the chest was rapidly opened and the heart exposed. Its movements remained regular. From the sudden ingress of air into the cavity of the chest, the left lung collapsed, and it was found necessary, in order to prevent asphyxia, to make artificial respirations on the right side of the chest. The pericardium having been opened, the right ventricle was punctured with the needle of an aspirator, and air injected in sufficient quantity to distend the right chambers of the heart, which immediately ceased to contract. On emptying the right ventricle by withdrawing the air and blood, the rhythmic movements of the organ were restored. These manipulations were repeated at least half a dozen times, and always with the same uniform and constant results. Half an hour after opening the chest all movements of the heart ceased. The heart was now removed and immersed in water at a temperature of 105° F., when it again commenced to pulsate at the rate of 120 beats per minute. After the lapse of about two minutes it was removed from the warm water, still pulsating, and transferred to cold water, when all movements ceased instantaneously, and all efforts to revive them proved futile.

Experiment No. 2.—Large Maltese cat. Heart exposed, right ventricle punctured, and 11 c. cm. of air injected with the result of

immediately arresting the pulsations of the right ventricle. When the air was withdrawn, the regular movements were renewed. This experiment was repeated on both ventricles eight or ten times in the course of half an hour with the same results. The auricles during all this time continued to contract. Ten minutes after the last puncture and evacuation the ventricles were still pulsating.

Remarks.—Both of these observations support the statement that sudden over-distension of any of the cavities of the heart by inflation with air, will arrest its movements in the diastole, and that upon the removal of the increased intra-cardiac pressure, the movements of the organ are restored. It was found that by continuing the injecting force after the right ventricle had become distended, air could be forced readily through the pulmonary capillaries into the left side of the heart. In some instances one of the cavities of the heart was kept over-distended and at rest for at least a minute, and yet on removing its contents the rhythmic movements were resumed.

XIV. *Aspiration of Right Ventricle for Air-Embolism.*

Being satisfied of the fact that sudden over-distension of the right ventricle may and does produce paralysis in the diastole, it appeared reasonable to resort to some direct measure in removing the cause of over-distension in grave cases of venous air-embolism. Puncturing and aspiration of the right ventricle seemed to offer the best chances for accomplishing this object. In the following experiments artificial venous air-embolism was produced in the usual way, when the right ventricle was punctured with the needle of an aspirator two millimetres in diameter, and the contents of the right side of the heart removed by aspiration. The region over the heart was shaved and disinfected, and the exact point of puncture located before the animal was anæsthetized. The needle was always carefully disinfected by passing it through the flame of an alcohol lamp, and immersion in carbolized water. When the puncture was made, the needle was advanced first only sufficiently deep to bury the opening in its point beneath the tissues, when a vacuum was created

in the aspirator so that the entrance of the needle into the ventricle would be promptly announced by the escape of spumous blood. By following this precaution, additional injury to the heart, by pushing the needle further into the cavity of the ventricle, was avoided, and at the same time a prompt escape of blood was secured. Experience taught me that it was very important not to push the needle too deeply into the cavity of the ventricle; not only for fear of inflicting additional injury to the endocardial lining opposite the point of entrance, but more particularly with a view of removing the free air which would naturally occupy the highest point in the cavity, if it was not intimately mixed with the blood. The needle was always directed obliquely from below upwards for the twofold purpose of making a valvular wound in the ventricle and of avoiding unnecessary injury to the endocardium.

Experiment No. 1.—Dog, weight 31 pounds. 60 c. cm. of air were injected into the left jugular vein. The animal was seized immediately with a convulsion, followed by collapse. Heart's action tumultuous. Churning sounds over heart were distinctly perceptible. Respirations rapid and superficial. Right ventricle was punctured, and 120 c. cm. of spumous blood were withdrawn. Pulsations of heart became more feeble, and as respirations had ceased and could not be restored, the chest was opened. The right ventricle was found moderately dilated and still contracting. Slight contractions of lower portion of left auricle. Left ventricle contracted, but without motion. The needle of the aspirator was then introduced into the right auricle, which was distended and tympanitic. After emptying it, it commenced to contract. All contractions ceased half an hour after the air was introduced. Faradic currents had no effect upon the heart after it had ceased to contract. The pericardium contained air in considerable quantity and about four c. cm. of fluid blood. Air was found in the right side of the heart, the left ventricle, coronary veins, and in the large veins in the immediate vicinity of the heart. Puncture about an inch from septum, and nearly equidistant between the apex and base of heart. No injury of endocardium on opposite side.

Experiment No. 2.—Sheep, weight 95 pounds. Before operation respirations 86, pulse 140. Injected 150 c. cm. of air into left

jugular vein, which was immediately followed by convulsions and involuntary discharges. Aspiration of right ventricle and removal of 150 c. cm. of spumous blood. As the animal still presented an asphyxiated appearance, the ligature was removed from the distal end of the jugular vein, and about 120 c. cm. of blood were allowed to escape. The animal showed no signs of improvement, and died five minutes after the injection was made, having made only five attempts at respiration during the time. Pericardium contained a small coagulum and a few bubbles of air. Right ventricle, distended and tympanitic, contained spumous blood. Two punctures were found over the middle of the right ventricle about one-half cm. apart; the second was made during the convulsive movements of the animal, which necessitated a partial withdrawal and reintroduction of the needle. On the inner surface each puncture was hermetically sealed by a minute thrombus which projected only very slightly into the cavity of the ventricle. No injury of opposite wall of ventricle. In the left ventricle, which was firmly contracted and empty, a small filiform coagulum projected through the aortic orifice into the aorta.

Experiment No. 3.—Old dog, weight 40 pounds. Injected 45 c. cm. of air into left jugular vein. Churning sounds over heart loud and distinct. Heart's action labored; respirations exceedingly rapid. No convulsion, but profound coma and involuntary discharges. A little more than one minute elapsed between the introduction of air and aspiration, during which time the heart's action became more and more embarrassed, and all of the symptoms became so grave, that death seemed unavoidable. But as the needle reached the heart, a slight impulse was imparted to it by the feeble pulsations, and 120 c cm. of air and spumous blood were removed. The heart's action improved immediately, but the dyspnœa, although less severe, was still quite prominent. Consequently an additional 120 c. cm. of blood were allowed to flow from the peripheral extremity of the jugular vein, which promptly relieved the urgent symptoms. Ten minutes after the injection, respirations were 240 per minute; four minutes later 360, and after eight minutes more 120. After forty minutes the stupor disappeared and the animal was able to walk. One hour after the operation, respirations were 20 and pulse 160 per minute. At this time the animal

walked home—a distance of nearly two miles—and subsequently suffered no inconvenience from the operation.

Experiment No. 4.—Dog, weight 75 pounds. Injected into left jugular vein 90 c. cm. of air, which produced intense difficulty in breathing and profound stupor. Sensation and reflex movements suspended. Heart's action exceedingly feeble and rapid. Churning sounds distinct. The aspirator and needle were kept in readiness to be used promptly on the approach of symptoms indicative of impending death. Two minutes after the injection the animal was apparently in a dying condition, when the needle was plunged into the right ventricle, and from its rapid oscillations it was apparent that the heart's action had not entirely ceased, but was very rapid and feeble. As quickly as possible 180 c. cm. of spumous blood were withdrawn, which promptly relieved the most urgent symptoms; but as the respirations still remained rapid and labored, the ligature was removed from the distal end of the jugular vein, and 240 c. cm. of blood were allowed to escape. Eight minutes later after the injection, the churning sounds over the heart were still present, but faint. Respirations 144; pulse 130. When the animal had recovered from the immediate effects of the operation, it was noticed that the posterior extremities could be moved only with difficulty, the gait being slow and staggering. For twelve hours the animal was dull and stupid, remaining in the same place without change of position. The drowsiness continued for thirty-six hours, after which the animal became lively, and went on to complete recovery without any further interruption. In this case it was apparent that the animal would have died from the large volume of air which had been injected, but for timely use of the needle and aspirator. Three weeks after the experiment the animal, being in perfect health, was killed by an arterial injection of air. No signs of pericarditis, endocarditis, or myocarditis. Puncture in ventricle indicated by a minute faint cicatrix.

Experiment No. 5.—Old dog, weight 25 pounds. In this case the aspirator was used a few seconds after the insufflation of air was made, and the result shows how important it is to withdraw the air from the ventricle before it has had time to escape into the pulmonary artery. Immediately after the injection of 90 c. cm. of air into the left jugular vein, the animal became comatose. Respiration

exceedingly rapid and heart's action imperceptible. The right ventricle was punctured at once and as soon as a part of its contents had been removed the impulse of the heart was imparted to the needle which could be distinctly seen and felt. Spumous blood and air to the amount of 150 c. cm. were withdrawn. The dog rallied rapidly after the aspiration, being able to walk in less than half an hour. He manifested no pain or discomfort from the air-embolism or operation. The volume of air in this case was three times as large as in the previous experiment, when the weight of the animals is compared. The immediate effects of the air were expended upon the heart as was made evident by the temporary cessation of its pulsations, and yet death was prevented by the prompt removal of the excessive intra-cardiac pressure. The recovery was more rapid than in the previous cases, from the fact that not so much air had passed into the pulmonary artery and more was removed by aspiration. Two weeks later, the animal being in perfect health, 60 c. cm. of air were injected into the right jugular vein, which produced death in a few minutes, showing conclusively that the recovery after the first insufflation was due to the aspiration. Cicatrix of puncture plainly visible. No signs of inflammation in the pericardium, endocardium, or substance of the heart.

Experiment No. 6.—Young dog, weight 28 pounds. In this experiment 120 c. cm. of air were thrown into the left jugular vein in divided doses of 30 c. cm. each in rapid succession. During the third injection the animal howled and became comatose, and immediately after the last injection the heart's action ceased and respirations were reduced to a few irregular gasps. The right ventricle was punctured and 120 c. cm. of spumous blood and air were withdrawn, without producing any effect upon the heart. Artificial respiration was resorted to without any better result. The chest was opened at once. The pericardium contained 4 c. cm. of dark, venous blood and a few air-bubbles. Right auricle continued to contract forty-eight times per minute. Left auricle and ventricle were distended and contained spumous blood. The needle had punctured the right auricle instead of the ventricle. All muscles, voluntary and involuntary, reacted promptly to the faradic current, but it had not the slightest effect upon the heart.

Experiment No. 7.—Young dog, weight 10 pounds. Injected 45 c. cm. of air into the left jugular vein. At the end of injection

the dog howled, and the heart's action ceased almost instantaneously. The right ventricle was aspirated and 60 c. cm. of spumous blood were withdrawn. Artificial respiration was performed and the faradic current was applied, when the dog made a few ineffectual attempts at respiration before he died. On examination the pericardium was found to contain a slight amount of dark fluid blood and a few air-bubbles. Both auricles contracted about forty-eight times in a minute. Right ventricle arrested in diastole. Puncture was found near base of ventricle. Right ventricle was again aspirated, and the faradic current was applied directly to the heart and the needle, without producing the slightest effect. Right ventricle and pulmonary artery contained spumous blood. Left ventricle contracted and nearly empty.

Experiment No. 8.—Adult dog, weight 24 pounds. Before operation, respirations 80; pulse 100 in a minute. Injection of 30 c. cm. of air into left jugular vein. Immediately after injection great restlessness, dyspnœa, and tumultuous action of heart. Churning sounds loud and distinct. Removed 90 c. cm. of spumous blood from right ventricle by aspiration. After the aspiration the improvement in respiration and pulse was marked. Recovery complete, and not attended by any complications.

Experiment No. 9.—Same animal as in experiment No. 3. Animal in perfect health; time since first operation three weeks. Injection of 40 c. cm. of air into right jugular vein. Symptoms the same as before. At the end of three minutes and a half the pulsations of the heart ceased, and the respirations were nearly suspended. The right ventricle was now punctured and 250 c. cm. of spumous blood were removed. No motion imparted to needle. Faradization and artificial respiration were ineffectual in restoring either the heart's action or respiration. On examination 30 c. cm. of dark fluid blood were found in the pericardium. No evidences of inflammation from former puncture. Right ventricle moderately distended with a clot of spumous blood. Point of second puncture one-half inch from coronary artery. The location of first puncture was marked near the base of the right ventricle by a faint minute cicatrix upon the pericardial surface, while the endocardium at a corresponding point showed an old circumscribed spot of ecchymosis, but no evidences of inflammation. Left ventricle contained no air and

only a minute quantity of fluid blood. Both auricles nearly empty. Pulmonary artery contained air and spumous blood. This experiment demonstrates positively and conclusively the value of early aspiration of the right ventricle in venous air-embolism, where death is threatened by over-distension of the right side of the heart, or by asphyxia. In the former experiment a larger amount of air was injected, but the animal was saved by an early aspiration by which a large amount of air was removed before it had time to accumulate in the pulmonary artery. It also illustrates the utter uselessness of resorting to any kind of mechanical interference after a fatal dose of air has once passed beyond the semilunar valves into the pulmonary artery. In such instances the air has passed beyond our reach, and will inevitably lead to a fatal result by asphyxia.

Experiment No. 10.—Adult dog, weight 18 pounds. Injection of 60 c. cm. of air into left jugular vein. Immediate collapse; churning sounds distinct. Aspiration of 100 c. cm. of air and spumous blood from right ventricle. Three minutes after the insufflation, respiration, and heart's action had ceased. No air or blood found in the pericardium. Needle puncture equidistant between base and apex of right ventricle, and about two cm. from septum. Right auricle contracting feebly forty times a minute. Right ventricle dilated, fibrillary contractions at apex only; right ventricle and pulmonary artery contained spumous blood. Left auricle and ventricle contained only a slight amount of fluid blood and a few air bubbles.

Remarks.—These experiments serve to illustrate the following interesting points in pathology and surgery: 1. The heart can be punctured with a perfectly aseptic, medium-sized needle of an aspirator, without any great immediate or remote danger. 2. Aspiration of the right ventricle for venous air-embolism, when done early enough (before a fatal dose of air has been forced into the pulmonary artery), must be considered in the light of a life-saving operation.

Although the needle used in all of these experiments was two millimetres in diameter, the hemorrhage into the pericardium was never sufficient in quantity to prove a source of danger. The largest quantity found was 30 c. cm. (in experiment No. 9).

In a number of cases no trace of blood was found in the pericardium. Against my expectations the same observations were made in regard to air. In one case the pericardium contained about 60 c. cm. of air, while in most cases only a few isolated air-bubbles were seen, while in others no trace of it could be found. In all cases where the animal survived the operation and was killed from a few days to three weeks subsequently, no trace of inflammation could be found either in serous membranes, muscular tissue, or adjacent organs. Even adhesion between the parietal and visceral layers of the pericardium at the point of puncture never occurred. The point of puncture was usually marked by a faint minute cicatrix in the visceral pericardium, unaccompanied by any pathological changes in the substance of the heart. We must take it for granted that if effusion of blood or air occurred into the pericardium in the animals which recovered, these adventitious substances caused no irritation, but were promptly removed by absorption.

With the exception of the subject of experiment No. 8, it may be safely assumed, that all of the animals would have died had aspiration not been performed, so that the operation saved at least three of the animals out of ten, or the whole number of experiments. The most conclusive proof of this statement was furnished by the subjects of experiments Nos. 3 and 5, as these animals died during subsequent experiments from the introduction of a much smaller quantity of air. The question naturally arises, Why were not all of the animals saved by the aspiration? In reply it may be stated that the amount of air injected in most instances was large, more than was necessary to produce a fatal result. Another element of failure consisted in the postponement of the aspiration until a fatal dose of air had passed beyond the reach of the aspirator. To prevent death by air-embolism it is essential to remove the air from the right ventricle as soon as possible after its entrance, before a fatal embolism of the pulmonary artery has had time to take place. In some of the experiments a fatal result might have been probably prevented by removing a larger quantity of air and blood with

the aspirator, as in some instances the condition of the animal was improved by a subsequent venesection from the jugular vein.

XV. *Catheterization and Aspiration of Right Auricle for Venous Air-Embolism.*

In grave cases of accidental air-embolism it would be a desideratum to be in possession of some means, by which the air could be removed directly from the right side of the heart in the shortest possible space of time and with the simplest instrument. An aspirator is not always at hand, and less frequently in a proper condition to be used on such short notice. It appeared to me that inasmuch as the accidental introduction of air usually takes place through wounds in one of the jugular veins, a catheter might be introduced through the wound into the vein and from there passed directly into the heart, and through it the air and spumous blood could be withdrawn by aspiration. A catheter is almost always at hand, and its introduction would require only a few moments of time. The following experiments were made to test the feasibility of this procedure.

Experiment No 1.—Adult dog, weight 24 pounds. The right jugular vein was opened and 60 c. cm. of air were introduced in the usual way. The breathing became very difficult and the heart's action labored. The animal was comatose, pupils dilated. As soon as it could be done, a number six, English scale, gum elastic catheter, which had previously been made aseptic, was introduced into the wound, and passed into the heart—a distance of 15 cm.—and with an exhausting syringe 250 c. cm. of air and spumous blood were removed. The animal recovered rapidly from the immediate effects of the air-embolism and operation, and subsequently manifested no symptoms of disease.

Experiment No. 2.—Young dog, weight 30 pounds. Injected 100 c. cm. of air into left jugular vein. The heart's action ceased almost instantaneously. The same catheter was introduced and pushed forward a distance of 18 cm. and about 120 c. cm. of air and spumous blood were withdrawn by the mouth. At this time

respiration ceased, and as no pulsations of the heart could be felt, the chest was opened at once. Fibrillary contractions of right ventricle which was distended and tympanitic. Left auricle and ventricle contracted and motionless. Right auricle contracted regularly 80 times a minute. Faradic and galvanic currents had no effect on ventricles. Right auricle responded to a galvanic current slowly interrupted. The catheter was again introduced, and its course observed as it entered the right auricle. It was found impossible by any manipulation to pass it from the auricle into the ventricle. In every instance it passed from the right auricle directly into the inferior vena cava. The right side of the heart was filled with water to test the possibility of removing fluids from the right auricle without introduction of the catheter into the chamber. A catheter with an open extremity was used. No amount of suction force succeeded in removing any of the fluid, until the open end of the catheter reached the auricle, when both chambers could be readily emptied. As long as the end of the catheter remained in the vein, the walls of the vein in front of the catheter would invariably collapse and close the opening in the catheter completely on applying suction force. This satisfied me that in order to remove air or blood from the right side of the heart, it is necessary to introduce the catheter as far as the auricle. It was also evident that in case the catheter be introduced too far, its distal end would pass into the inferior vena cava, and on aspiration only venous blood would be withdrawn, not air and spumous blood from the right chambers of the heart, for the removal of which the procedure was intended. On opening the right side of the heart, a large spongy clot of spumous blood was found in the right auricle, which extended for some distance into the ventricle. Left ventricle contained only a small amount of fluid blood and a few air-bubbles. As the heart's action had ceased before catheterization, death was undoubtedly due to the insufflation of air and not to the formation of a thrombus in the heart.

Experiment No. 3.—Adult dog, weight 20 pounds. Injected 30 c. cm. of air into right jugular vein. Catheterization and aspiration of right auricle, by which 90 c. cm. of air and spumous blood were removed when catheter became impermeable by a thrombus in its interior and was removed. Before emptying the right side of the heart the animal was comatose, breathing and heart's action exceedingly rapid. After aspiration rapid improvement and complete re-

covery. If the catheter in this instance had been permitted to remain longer in the auricle, the thrombus which formed in the interior of the instrument would undoubtedly have increased rapidly, and would have extended to the auricle, in which event death from thrombosis would have been unavoidable.

Experiment No 4.—Old dog, weight 40 pounds. Before operation respirations 16; pulse 100 per minute. Injection of 90 c. cm. of air into left jugular vein. Passed a No. 5 Nélaton's catheter into right auricle, a distance of 27 cm., and removed 250 c. cm. of air and spumous blood. Immediately after the insufflation the animal uttered a howl and became comatose. Pulse 250 per minute, respirations so rapid that they could not be counted. Seven minutes after catheterization heart's action very irregular, tumultuous, and about 300 beats per minute. Two minutes later respiration ceased, after which the heart's action became slow and feeble, only to cease a few seconds later. Internal mammary and coronary veins filled with air. Left side of heart contained a small amount of bright red, frothy blood. Right ventricle moderately dilated, showed fibrillary contractions. Superior and inferior vena cava, right auricle and ventricle contained a continuous, soft thrombus, the oldest portion of which corresponded to the superior vena cava. The upper portion of the clot in the inferior vena cava contained air. Death in this instance was due to the formation of a thrombus.

Experiment No. 5.—Adult dog, weight 25 pounds. Injection of 60 c. cm. of air into left jugular vein produced immediate collapse, distressing dyspnœa, and great rapidity and irregularity of heart's action. The dog howled when the heart's action ceased, and respiration became gasping. Some delay was experienced in the introduction of the catheter, and when the instrument was in place all signs of life had disappeared. About 50 c. cm. of spumous blood were withdrawn without any signs of improvement. Artificial respiration and faradization were resorted to without producing the slightest impression. On opening the chest it was observed that the right auricle contracted about 30 times a minute. Irregular fibrillary contractions of apex of right ventricle. Right side of heart moderately distended with spumous blood. No thrombus. Left ventricle almost empty.

Experiment No. 6.—Adult dog, weight 15 pounds. Injection of 60 c. cm. of air into right jugular vein. In this case the catheter was connected with a rubber-tube three feet in length, which was kept under water; then the instrument was pushed along the jugular vein into the right auricle. The injection of air was followed by the most urgent and distressing symptoms. Only a small amount of dark venous blood escaped through the rubber-tube until the catheter reached the right auricle, when a gush of spumous blood escaped from the tube under water. As the blood did not continue to escape, about 60 c. cm. of spumous blood were withdrawn. After the aspiration the symptoms improved rapidly, and within twenty-five minutes, with the exception of a slight excess in the respiratory movements, the animal appeared to be perfectly well. The ultimate recovery was complete and not disturbed by any complications.

Experiment No. 7.—Adult dog, weight 48 pounds. Before operation pulse 150; respirations 28. Injection of 120 c. cm. of air into right jugular vein. The animal howled and collapsed. Respirations increased at once to 100 per minute. Heart's action tumultuous, and churning sounds loud and distinct. Passed a Nélaton's catheter into the right auricle, and aspirated 360 c. cm. of air and spumous blood. No signs of improvement followed, and in fifteen minutes the animal was dead. At the post-mortem examination the internal mammary veins were found distended with air. Right side of heart tympanitic and distended. Slight fibrillary contractions of right auricle. Apex of right ventricle contracted regularly 22 times in a minute. Coronary veins filled with air. A thrombus had formed, which extended from the superior vena cava into right auricle, ventricle, and for a distance of 25 cm. into the inferior vena cava. Pulmonary artery contained no thrombus, but was distended with air and spumous blood. Left ventricle contracted, contained a small amount of fluid blood and air-bubbles. It was quite evident that the thrombus had primarily formed around the catheter, and by new additions it finally reached the distal portion of the inferior vena cava. Death was produced by thrombosis.

Remarks.—The therapeutical value of catheterization and aspiration of the right auricle for air-embolism is made apparent by the following tabular arrangement of the experiments:—

No. of experiment.	Weight of animal.	Amount of air injected.	Amount of air and blood removed.	Result.	Cause of death.
1	24 pounds.	60 c. cm.	250 c. cm.	Recovery.	
2	30 "	100 "	120 "	Death	Thrombosis.
3	20 "	30 "	90 "	Recovery.	
4	40 "	90 "	250 "	Death	Thrombosis.
5	25 "	60 "	50 "	"	Air-embolism.
6	15 "	60 "	60 "	Recovery.	
7	48 "	120 "	360 "	Death	Thrombosis.

From this table it will be seen that of seven animals subjected to the operation, three recovered and four died. The amount of air injected in the subjects of experiments Nos. 1 and 6 was sufficient to destroy life, so that we can safely assume that the animals would have died, but for the speedy resort to catheterization and aspiration. In the subject of experiment No. 3 recovery might have taken place without the operation, as the relative quantity of air was much smaller. In the four fatal cases death was produced only once from the presence of air, while the direct cause of death in the remaining three was due to thrombosis. All possible precautions were exercised to prevent the accident. The catheter was made aseptic by thorough cleansing and immersion in a five per cent. solution of carbolic acid. Before the operation the instrument was kept ready for use in an alkaline solution of the temperature of the body, and yet this accident happened in three out of seven cases. It is now asserted that the introduction of aseptic bodies into the circulation does not give rise to thrombosis, and it may be possible that some of the instruments used, in spite of the pains taken to render them aseptic, were still not in a fit condition to be used for such a purpose. Catheterization and aspiration of the right auricle for air-embolism compare favorably with puncture and aspiration of the right ventricle as a life-saving procedure, but the former operation is more dangerous on account of the tendency to the formation of a thrombus within or around the catheter. If this formation of thrombus could be avoided with certainty, catheterization and aspiration of the right auricle

would recommend itself as the most expedient and reliable therapeutic agent in cases where life is threatened by air-embolism.

XVI. *Prophylactic Treatment of Air-Embolism.*

As clinical experience and experimental research have shown that the admission of air into veins is not an infrequent occurrence and often has resulted in death, it is the duty of the surgeon in extirpating tumors, which are in close proximity to the large veins within the area of the "danger zone" to resort to measures which will prevent the ingress of air in case a vein is accidentally wounded. The following precautionary means deserve consideration: 1. Position. 2. Compression. 3. Ligature. 4. Aseptic tampon.

1. *Position.*—Regular respiratory movements of the chest are necessary to maintain the equilibrium between the arterial and venous circulation, and should always be secured in operating in close proximity to veins at the base of the neck, as during a sudden deep inspiration the direct aspiratory effect of respiration in the veins extends to some distance, constituting thus the direct cause of entrance of air into an open vein. Before anæsthetics were used, Poiseuille advised that the patient should be instructed not to make any deep inspiratory movements during the operation. At the present time the same object is better obtained by keeping the patient thoroughly and continuously under the influence of the anæsthetic. Gerdy aimed to prevent the aspiratory effect of respiration by recommending compression of the thorax, but the advice is more useful in theory than in practice, as suspension of the thoracic movements for any length of time would necessarily interfere with respiration and cause death by asphyxia.

The venous circulation is greatly influenced by position. In the elevated position the blood gravitates towards the heart and the veins are emptied. If in this position a vein is wounded, and the walls of the vessel do not collapse on account of some anatomical peculiarities, or from rigidity of the vein wall itself,

due to pathological changes, a vacuum is formed and air enters. The entrance of air into a vein distended with blood is a physical impossibility. In reading the clinical histories of cases where air entered veins during operations, we are met by the fact that in almost all of the cases, the wounding of the vein was not followed by any considerable loss of blood, and we are usually told that the air entered almost immediately after the vessel was injured, which would indicate that the vein was empty or nearly so at the time it was wounded. In a number of cases the bloodless condition of the vein attracted the attention of the operator, and is particularly emphasized in the description of the cases. As long as the interior of a vein and the lumen of a wound in its walls are occupied by a continuous stream of blood there is no danger that air will be admitted. Statistics tend to show that the accidental admission of air into veins was more frequent before anæsthetics were used, a fact which we can only explain by assuming that the patients were then usually placed in a sitting or semi-recumbent position during the operation, positions favorable to the return of venous blood from the cervical region. On the other hand, the safe administration of an anæsthetic necessitates the horizontal position in which the veins of the neck become distended with blood. In the former instance the return of venous blood in the vessels of the upper portion of the body was favored by the elevated position, and the vacuity thus produced in the veins constituted the most important and potent factor in determining the entrance of air. In the horizontal position, the veins of the neck are never empty, and to effect entrance of air the exciting cause must operate with increased intensity. On this account the horizontal or dependent position of the region of the body to be operated upon, recommends itself as the simplest prophylactic measure against the accidental entrance of air into wounded veins. This position increases the risk of hemorrhage from the injured vein, but this accident in its immediate effects is less disastrous to the patient and more readily under the control of the surgeon than air-embolism.

2. *Compression.*—Compression of a vein between the seat of

operation and the heart for the purpose of guarding against the entrance of air may be divided into intermediate and direct. Intermediate compression is only applicable in cases where the external jugular vein is in danger of being wounded. If the tumor to be extirpated is not located too low in the region of the neck uninterrupted digital compression of the external jugular vein just above the clavicle can be relied upon in preventing entrance of air through this vessel. The internal jugular and axillary veins are so deeply situated that intermediate digital compression cannot be relied upon in the prevention of air-embolism, consequently it becomes necessary to resort to direct compression whenever it becomes necessary to guard against this accident. Warren recommended that in the extirpation of tumors which are in close proximity to the large veins of the neck and axilla, the separation of the pedicle should be reserved until the last in order to enable the operator to compress the vein between the seat of operation and the heart more effectually, should the vessel be injured during the operation.

Langenbeck advised that the vein on the distal and proximal sides of the tumor should be exposed to sight as a preliminary measure before attempting the removal of the tumor with a view to facilitate direct compression in case the vessel should be opened during the dissection. The adoption of either of these precautions would successfully prevent a second ingress of air, but would not protect the patient against the dangers arising from the first dose. Permanent central compression augments the danger of wounding the vein from the turgidity of the vessel which it produces, while distal compression would be attended by the same risk, only in an opposite way, by rendering the vessel empty and consequently more difficult of recognition. If the attachments of the tumor to the vein are of such a nature that they can be separated without great danger of injuring the vessel, then the vein should be isolated on the proximal side and the vessel compressed with a hæmostatic forceps which will afford greater safety than digital compression, at the same time the field of operation is not obscured and narrowed by the

fingers and hand of the assistant, an item of great importance in operating in the regions of the neck or axillary space.

Injury of the vein in such instances will be promptly announced by hemorrhage from the distal portion, but the entrance of air into the right ventricle is made impossible by the prophylactic compression of the vessel between the seat of operation and the heart. Should the vein be wounded, any further ill effects arising from this accident can be prevented by applying a lateral ligature or tying both ends of the vein according to the size of the wound, before removing the compressing forceps. In all operations where any of the large veins are in danger of being wounded the position of the parts should be such as to retain as nearly as possible their normal anatomical relations. The operator should make haste slowly, and identify every structure before using cutting instruments. In extirpating tumors that are deeply situated, the external incision should always be large so as to afford free access to the base or deepest portion of the growth to be removed. A pair of Billroth's retractors will do good service during the deep dissection.

Hemorrhage must be carefully arrested as it occurs, in order to enable the surgeon to see what he is doing. Isolation of the vein from the tumor must be accomplished by the use of blunt instruments, and all firmer attachments should be carefully examined and identified before using the knife or scissors. As it is usually impossible, or, at any rate, impracticable to find the vein in the tumor or its immediate vicinity, the rule should always be followed to expose the vessel for some distance from the tumor on the proximate side, and then follow it by carefully separating the attached tissues by means of blunt instruments. Under antiseptic precautions the jugular vein can be isolated for a great distance without fear of compromising the vitality of its tissues or causing thrombosis. When a tumor is attached to the jugular vein it is a dangerous practice to make traction on the tumor for the purpose of facilitating the deep dissection, as this procedure will disturb the normal anatomical relations of the vessel and thus expose it to greater risk of being wounded. Free access to the base of the tumor must be secured without

displacing the subjacent vein. As soon as the tumor has been isolated from the surrounding tissues a thorough examination should be made of the extent of its base and its relations to the subjacent vein before proceeding any further with the dissection. If the conclusion is reached that the vein is so intimately connected with the base of the tumor that it is in great danger of being injured during the further progress of the extirpation, it becomes necessary to resort to direct compression of the distal and proximal portion of the vein. By isolating and compressing the vein on each side of the tumor both excessive turgidity and emptiness of the intervening portion of the vein are avoided. The amount of blood in the vein between the two points of compression can be regulated, and only a sufficient quantity is allowed to remain to indicate the exact location of the vessel. Compression is again made with hæmostatic forceps. After the complete removal of the tumor the forceps on the distal side of the vein is removed first, in order to test the integrity of the vessel and with a view to prevent any possibility of the entrance of air. If no hemorrhage takes place we may be satisfied that the vein has not been injured, and the remaining pair of forceps can be safely removed. In such cases the exposed portion of the vein should always be covered with the adjacent tissues by deep sutures *en étages* of catgut before closing the wound. If the vein has been wounded a lateral ligature is applied, or both ends are ligated after complete division of the vessel before the forceps between the vein wound and the heart is removed. By following this plan we accomplish the following objects: 1. Hemorrhage from the injured vein is slight and perfectly under the control of the surgeon. 2. Absolute security against the entrance of air. 3. A more thorough removal of the tumor.

3. *Ligature.*—In all cases where a large vein passes through the substance of the tumor and where isolation of the vessel appears inexpedient or impossible, double ligation recommends itself as the safest prophylactic measure both against hemorrhage and air-embolism. As soon as the base of the tumor is reached the vein is exposed on each side and tied with catgut, and the

intervening portion is extirpated with the tumor. By adopting this plan we secure for the patient absolute protection against hemorrhage and air-embolism. Adequate collateral circulation is established in a short time, so that ligation or excision of the internal jugular or axillary veins can be done under antiseptic precautions without danger of causing serious disturbance of the cerebral circulation in the former, or gangrene of the arm in the latter instance. Isolation and ligation of the vein on the proximal side of the tumor should always be resorted to, when injury to the vein is unavoidable. In case the prophylactic ligation of the vein on the distal side of the tumor is rendered difficult or impossible by existing circumstances, the following course can be pursued. After ligation of the vein on the proximal side of the tumor a hæmostatic forceps is applied a little distance from the ligature on the side of the tumor, and the vessel is divided between the forceps and the ligature. The ligature prevents the entrance of air, and the forceps serves the purpose of a temporary hæmostatic agent during the extirpation of the tumor. The base of the tumor with the vein is detached when the vessel on the distal side of the tumor is sought for and tied, and the remaining attachments of the tumor, including the vein, are cut. This plan affords protection against the entrance of air, but during the separation of the tumor hemorrhage may occur from accidental wounds of the distal portion of the vein, which, however, can be readily controlled by compression with hæmostatic forceps until the vessel is permanently secured on the distal side of the tumor.

4. *Aseptic Tampon.*—If a large vein has been wounded which from its location is inaccessible to the ligature, permanent compression with a graduated aseptic tampon will arrest the hemorrhage and prevent further ingress of air.

XVII. *Operative Treatment of Air-Embolism.*

Considering the infrequency of air-embolism as compared with the number of vein wounds, it is not surprising to find that, as a rule, surgical writers have little, if anything, to say on the

subject of the prevention and treatment of air-embolism. It must be apparent to all, that in following the rules laid down when we considered the prophylactic treatment, the accidental introduction of air into veins is prevented almost to a certainty, and at the same time they will also furnish a safe guide in the prevention and management of venous hemorrhage when operating within the area of the "danger zone." If the preventive treatment is carefully carried out, air-embolism will become one of the rarest accidents in surgery. As even with the greatest care an accident of this kind might happen, it becomes necessary to allude to the operative treatment of air-embolism. The occurrence of the accident is sometimes announced by an audible hissing or sucking sound, and is always followed almost instantly by a well-marked train of distressing symptoms which are directly referable to a mechanical obstruction to the circulation in the right side of the heart and the pulmonary artery. The treatment must depend on the quantity of air which has entered, and the severity of the symptoms produced by it. The therapeutic measures should be aimed towards meeting the following indications: 1. Prevention of further ingress of air. 2. Administration of cardiac stimulants to sustain the action of the heart. 3. Venesection to relieve intravenous pressure. 4. Aspiration of air from the right side of the heart to prevent over-distension and paralysis of right ventricle.

1. When air has entered through a gaping wound in a vein during inspiration there is great danger that the same occurrence will repeat itself during successive respiratory movements of the chest, hence the first object of treatment consists in closing the wound in the vein. This is most quickly done by digital compression, which is continued until the vein has been rendered impermeable by ligature or permanent compression. If the symptoms are urgent no time should be lost in securing the vessel until the patient has rallied from the immediate effects of the air-embolism. It is also necessary to postpone the permanent closure of the vein until the time has elapsed for any indications to arise which would call for venesection or operative removal of the air from the right side of the heart, as in such

cases it may become necessary to utilize the wounded vein as a route for the introduction of a catheter into the heart. When the time has arrived for closing the vein the finger should not be removed suddenly from the vessel for fear of causing a repetition of the accident; it should remain *in situ* until the vessel can be compressed on the proximal side of the vein wound by an assistant. The exact location of the wound will now be probably indicated by escape of blood from the distal end of the vein when the vessel can be seized with a forceps, and after its isolation, a double ligature is applied. If it is found impossible to ligate the vein, then the vessel is compressed in the wound by means of a graduated aseptic tampon retained in place by uninterrupted pressure until definitive closure of the wound has taken place.

2. The heart's action must be supported by position and by the use of cardiac stimulants, which are best administered subcutaneously and by inhalation. The patient must be placed in the horizontal position with a view of guarding as much as possible against the occurrence of cerebral anæmia as well as to lessen the intravascular pressure to a minimum. If the quantity of air admitted is not sufficient to produce instant death by paralysis of the right side of the heart in the diastole, the symptoms which follow always point towards an embarrassment of the circulation in the right side of the heart and the pulmonary artery, which are always accompanied by dyspnœa, combined with evidences indicating the existence of acute cerebral ischæmia. If death does not take place within a few minutes from obstruction to the circulation through the pulmonary artery by the presence of air in that vessel, then the contractions of the right ventricle will force at least a part of the air through the pulmonary capillaries into the general circulation, thus preventing death by asphyxia. As soon as the air-embolism of the pulmonary artery has been relieved the most urgent symptoms subside, as the air-emboli in the arterial system are distributed over a larger area and consequently produce an embolism of lesser extent and gravity in more distant and less essential organs. The safety of the patient depends on the capacity of

the right ventricle to force the air through the pulmonary into the general circulation, in other words, upon the time which is required in removing the emboli from the pulmonary artery into the systemic circulation.

Nitrite of amyl, from its stimulating properties upon the heart and from the rapidity of its action, would recommend itself in the form of inhalation as the most efficient drug in preventing threatened syncope. The temporary dilatation of the small bloodvessels would tend to produce, at least for a short time, a diminution of the intravascular pressure. Hypodermatic injections of camphor and alcohol, although slower in their action, would also assist in antagonizing the deleterious effect of over-distension of the right ventricle by the air-embolus. In all of my experiments where electricity was used it had no effect whatever upon the heart, even when applied directly to the organ, hence it would be of no use to resort to this agent for the purpose of sustaining or re-establishing the movements of this organ in serious cases of air-embolism.

3. In all post-mortem examinations after sudden death from air-embolism two constant pathological conditions are always found present: 1. A comparatively empty state in the left side of the heart and throughout the entire arterial system. 2. Distension of the right ventricle and intense engorgement of the whole venous system. Both of these conditions are, of course, due to the presence of air in the ultimate branches of the pulmonary artery, which prevents the free transit of the venous blood through the pulmonary capillaries into the left side of the heart. As a direct result of these conditions, we have a diminution of the intravascular pressure in the left side of the heart and arteries, and a corresponding increase in the right side of the heart and the veins. The immediate and greatest danger arises from an accumulation of air and blood in the right side of the heart to such an extent as will arrest the ventricular contractions by over-distension before the equilibrium between the arterial and venous circulation can be restored by the removal of the air-emboli from the pulmonary artery into the general circulation. Even the most extreme antagonists to the lancet

must acknowledge the benefit which follows its use in similar conditions of the circulation caused by other pathological conditions. In severe cases of pneumonia where the circulation has been so much obstructed that death is threatened by venous engorgement and over-distension of the right ventricle, the free use of the lancet is the safest and most efficient means in equalizing the circulation, as by the abstraction of blood from one of the veins, the intravascular pressure in the veins and right side of the heart is diminished in the promptest and most direct manner. As the adventitious air in the pulmonary capillaries can and will be forced into the general circulation by the contractions of the right ventricle, and as the urgent symptoms will subside after this has taken place, it is of paramount importance to gain time by protecting the right ventricle against an undue amount of additional labor. Among the indirect remedies to accomplish this object venesection deserves the first place. I could refer to a number of the experiments where copious bleeding from the distal portion of the open vein was promptly followed by improvement in all the symptoms. Vulpian asserted that, after insufflation of air into veins, the contractions of the heart were restored after the organ had ceased pulsating, by the abstraction of blood from the sinuses of the brain and the veins of the neck. I have never succeeded in restoring the action of the heart, after its pulsations had ceased completely, by any kind of treatment; but, when the contractions have become imperfect from this cause, I know that removal of the cause of over-distension will restore the force and efficiency of the contractions. H. Fischer[1] sarcastically alludes to this valuable agent in the treatment of air-embolism as are medy from which only homœopaths would expect to derive any benefit. An expression of this kind concerning such a valuable remedy, and in this particular connection, must be looked upon as unscientific and contrary to well-established clinical and pathological facts. I must, therefore, insist that venesection from the distal end of the wounded vein will prove beneficial in all cases of air-embolism where venous

[1] Ueber die Gefahren des Lufteintritts in die Venen, p. 17.

engorgement and over-distension of the right ventricle constitute elements of imminent danger. If practicable, the bleeding should always be done from the distal end of the wounded vein while the proximal end is compressed or ligated. The advantages of doing so are obvious for the following reasons: *a.* The abstraction of blood is accomplished in the shortest possible space of time. *b.* The blood escapes in a large stream from a capacious vessel. *c.* No additional instruments are required, and the infliction of another wound is obviated.

4. Clinical experience and experimental research teach us that when a certain amount of air enters the right side of the heart, death invariably takes place in a very short time and cannot be prevented by any of the indirect methods of treatment. It seems to me that in such cases it would be legitimate and proper for the surgeon to resort to some procedure by which the air could be removed directly from the heart. To accomplish this object Fischer recommends that forcible expiratory movements should be excited by inducing coughing, sneezing, or vomiting, with a view that during the forcible compression of the thorax the aspirated air would be forced out through the wound in the vein. Against this advice the following objections may be entered: 1. The difficulty or impossibility of exciting coughing, sneezing, vomiting, or any other act on part of the patient when in a condition of collapse. 2. If during the forcible compression of the chest, the air is forced backwards, it will be just as likely to pass into other veins than the one through which it entered. 3. The difficulty of preventing the admission of more air during the forcible inspiration following the forcible expiration. 4. During prolonged forcible expiration the intravascular pressure in the veins and right side of the heart is greatly increased, thus constituting an additional source of immediate danger. 5. Forcible expiratory movements of the thorax, by compressing the heart, will be more likely to force the air onward with the venous current into the pulmonary artery where it will do the most harm by causing asphyxia from a sudden and extensive air-embolism in that vessel.

The only direct means of removing air from the heart consist

in puncture of the right ventricle and catheterization of the right auricle combined with aspiration. The experiments made by myself in this direction have demonstrated that puncture of the right ventricle with an aseptic-needle two millimetres in diameter is in itself a harmless procedure. When we remember that in the human subject the heart has often been the seat of more extensive injury without any immediate or remote ill effects, we must abandon the idea that slight injuries of this organ are necessarily fatal. Small aseptic wounds of the heart heal rapidly, and in the same manner as in any other organ of the body. The hearts which I show you were removed from dogs a few days to three weeks after puncture; and not in a single specimen are we able to detect any evidences of organic changes, either in the substance of the organ or its serous membranes. In most of the specimens the point of puncture is marked by a minute cicatrix, visible upon the surface of the visceral pericardium. In penetrating wounds of the heart hemorrhage into the pericardium and compression of the heart from this cause are to be feared, and constitute the only source of immediate danger. There is no plausible reason why in the human subject an oblique puncture of the right ventricle should be followed by more hemorrhage than in animals, and consequently I have no hesitation in recommending puncture and aspiration of the heart as a justifiable procedure in cases of air-embolism which would otherwise necessarily prove fatal. The question naturally arises: What symptoms indicate a resort to puncture and aspiration of the right ventricle? Two different conditions, as far as time and symptoms are concerned, may call for this operation. The puncture should be made as soon as possible after the entrance of air, in the event that the primary effect of the heart embolus has produced sudden over-distension and paralysis of the right ventricle, an occurrence which would be indicated by immediate collapse and partial or complete suspension of the heart's action. In such a case the direct withdrawal of air from the right ventricle, as soon as possible after its entrance, affords the only possible hope of restoring the pulsations of the heart by removing the cause of

the mechanical over-distension. In the second class of cases the patient has collapsed, but the heart has withstood the primary effect of the aspirated air. The heart's action is rapid and tumultuous, perhaps at times intermittent. The ear applied over the cardiac region detects distinct churning sounds. Respirations are rapid, and all symptoms point towards imperfect circulation and aëration of the blood as expressed by the pallid face and blue lips. Some of the air has passed from the right side of the heart into the pulmonary artery, and from the obstruction of the circulation through this vessel the right ventricle becomes more and more distended, the contractions therefore are less perfect and more frequent. At this stage of things puncture and aspiration of the right ventricle will overcome the most urgent symptoms by the removal of air and spumous blood, and enough time may be gained for the right ventricle to force the remaining air-emboli through the pulmonary capillaries into the general circulation, and the life of the patient is saved. These are the cases where puncture and aspiration of the right ventricle can be done with a fair prospect of not only relieving urgent symptoms, but of ultimate recovery. They are also more favorable from the fact that more time is afforded the surgeon in procuring and using the aspirator. The puncture should always be made in an oblique direction, from below upwards, so as to make a valvular tract in the heart for the purpose of preventing hemorrhage into the pericardium, and also for giving the point of the needle a direction in which it is least likely to injure the opposite endocardial lining. The left intercostal space, between the fourth and fifth ribs, about an inch and one-half from the margin of the sternum, is selected as the best point for making the puncture. The needle, thoroughly disinfected, is connected with the aspirator, and, as soon as its point is buried in the tissues, a vacuum is created in the aspirator, and the needle is advanced slowly until spumous blood is felt and seen to escape, when it is firmly held in this position, and the contents of the ventricle are withdrawn as quickly as possible. My experiments have satisfied me that I generally removed the needle too soon, not having withdrawn a

sufficient quantity of blood and air. In some of the experiments I followed the aspiration by blood-letting with marked benefit. Removal of the same quantity of blood directly from the right ventricle would have been productive of more good, as less air would have been left in the heart. The experiments on dogs have shown that these animals will always recover if the quantity of air injected into the veins does not exceed one c. cm. of air to each pound of its weight. Double this quantity must be considered a fatal dose. If, therefore, we can remove by aspiration only a portion of air directly from the right ventricle we may be able to maintain the action of the heart and respiration until the embolism of the pulmonary artery has been relieved by the passage of air from this vessel into the general circulation. When the presence of a foreign body in any other part of the body threatens to destroy life no surgeon would hesitate to make an attempt to remove it, even if the effort should be attended by an increase of the immediate risk. Air in the right side of the heart acts the part of a foreign body, and, when it destroys life, it does so by causing mechanical obstruction to the circulation. The timely removal of the air is the only rational treatment in all cases where simpler measures have proved inadequate in preventing a fatal termination. An aspirator in good condition should be on hand in every well-regulated hospital, and of ready access in cases of emergency. If air-embolism occurs during an operation the instrument should be used before the heart has ceased pulsating, when prompt action on part of the operator may obviate death in an otherwise hopeless case. The experiments on catheterization of the heart were undertaken with a view to simplify the operative removal of air from the right side of the heart. An aseptic catheter was introduced into the wounded vein and passed into the right auricle, and by aspiration air and spumous blood were removed. The result showed that some of the animals were saved from impending death by this simple procedure. The great danger attending this operation consisted in the tendency of the blood to coagulate within or around the distal end of the catheter, and death from the formation of a thrombus in the

large veins and right side of the heart. If the formation of a thrombus could in some way be prevented with certainty, catheterization and aspiration of the heart would recommend itself as the simplest and safest measure in the operative treatment of air-embolism.

Catheterization of the heart is not a new suggestion, as the introduction of a tube into the heart for the same purpose was recommended more than fifty years ago by Magendie. I should recommend the adoption of this procedure only in case when the air has entered through a wound in the internal jugular, and when the symptoms leave no doubt that death would be inevitable without direct removal of the air by aspiration. An aseptic Nélaton's catheter with an open end should be introduced into the wound, through which the air entered, and pushed as quickly as possible as far as the auricle, when the air and spumous blood can be withdrawn by the mouth in the absence of any other means of aspiration. Admission of more air is prevented by compression of the distal end of the instrument before and between the aspirations. The whole operation must be done as quickly as possible for the purpose of preventing coagulation of the blood. For the same reason an instrument of large calibre should be used.

In presenting to you the practical outcome of my experimental work in the form of these few suggestions on the operative treatment of air-embolism, I am fully aware that in your own minds you have decided that my labor has been in vain, and that the means suggested do not admit of application in practice. In reply, I will say that desperate cases call for desperate measures. When death stares us in the face we have not only a right, but it becomes our most imperative duty to resort to any plan of treatment which holds out the slightest hope of saving the life of the patient. For myself I am fully convinced of the safety and usefulness of puncture and aspiration of the right ventricle in grave cases of air-embolism, where simpler means have proved of no avail. When no aspirator is within reach I also believe in the propriety of catheterization and aspiration of the heart as a last resort in all cases of air-

embolism where death would surely take place without it. If by the adoption of either of these methods of direct treatment of air-embolism a single human life should be saved, I shall feel amply rewarded for the labor incurred in the preparation of this paper.

In conclusion, I beg leave to submit the following *résumé* for your further deliberation and discussion:—

1. The presence of adventitious air in the vascular system during life gives rise to air-embolism.

2. Each air-embolus constitutes a mechanical source of partial or complete obstruction to the flow of blood in the vessel in which it is located.

3. Aspiration during the inspiratory movements of the chest is the direct or exciting cause of ingress of air into a wounded vein or sinus.

4. Elevation of the head is the sole predisposing cause of the entrance of air in wounds of the superior longitudinal sinus.

5. In veins the predisposing causes consist in—
   a. Elevation of the part wounded.
   b. Pathological or anatomical conditions which prevent collapse of the vein when it is wounded.

6. Insufflation of a fatal quantity of air into a vein produces death by—
   a. Mechanical over-distension of the right ventricle of the heart, and paralysis in the diastole.
   b. Asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery.

7. Insufflation of the same quantity of air into arteries is less dangerous than when introduced into veins. When death is produced in this manner it results from—
   a. Acute cerebral ischæmia.
   b. Secondary venous air-embolism.
   c. Intense collateral engorgement of the vessels of the brain and spinal cord. The manner of death being

determined by the amount of air injected, and the direction in which the injection is thrown, as well as the time which has elapsed between the operation and the fatal termination.

8. Air injected into arteries is readily forced through the systemic capillaries into the venous circulation and right side of the heart by the powerful contractions of the left ventricle.

9. Air-embolism of the pulmonary artery is relieved in a comparatively short time, provided the contractions of the right ventricle continue unimpaired for a sufficient length of time to force the air through the pulmonary capillaries into the general circulation.

10. The prophylactic treatment consists in proximal or double compression, or ligation of the vein which is endangered by the operation.

11. The indirect treatment has for its objects—

a. Prevention of admission of air.

b. Administration by inhalation of hypodermatic injection of cardiac stimulants.

c. Venesection.

12. The direct or operative treatment by—

a. Puncture and aspiration of the right ventricle.

b. Catheterization and aspiration of right auricle, which are proposed with a view to obviate the direct cause of death by the removal of air and spumous blood, thus relieving directly the over-distension of the right ventricle, and, at the same time, to guard against a fatal embolism of the pulmonary artery.

13. The results obtained by experiments on animals warrant the adoption of the operative treatment of air-embolism in practice, as a last resort, in all cases where the indirect treatment has proved inadequate to meet the urgent indications.

DISCUSSION.

Dr. J. Collins Warren, of Boston, Massachusetts.

I do not know that it is necessary to say anything in regard to the admiration which we all feel for the arduous labor of Dr. Senn. The work is of a character that we should value and cherish. We have too little of this kind of work in this country, and should be glad to see more of it.

In regard to the lesion of which he treats, I think that the criticism may be made that it is rather obstetrical than surgical. There are few members of this Association who have met with such an accident that has called for any exercise of their surgical skill. A certain amount of aspiration of air into veins has been seen by most every surgeon, but alarming symptoms are of rare occurrence. The cases are not numerous in surgical literature.

According to the investigation of those who have studied this subject beside Dr. Senn, it appears that the lesion amounts to a blowing up of the right side of the heart, so that its action is paralyzed and it cannot contract. Various other theories as to the cause of death have been brought forward, such as anæmia of the brain and air-embolism of the lung, but it seems to me that the lesion described is sufficient to explain the result. The heart undertakes to force out of it that which it cannot squeeze or compress, and it is paralyzed in its efforts to do it. It has been compared by some to fat-embolism. We know that fat-embolism, which sometimes follows surgical injuries, may be present and not produce any symptoms. The capillaries of the lung may be filled with fat-emboli and yet no symptoms be produced. At autopsies a large amount of such deposits are sometimes found without their existence having been suspected during life. One lung may be completely obstructed by disease, and yet the patient be in a condition to live, as far as the lung itself is concerned. It does not seem probable that the presence of air in the lung is necessarily fatal. It is the heart and the large vessels which bring the blood to the heart that are blocked, and circulation in them is stopped.

Experiment has also shown that a large quantity of air can be injected into the circulation if it is done slowly. We have then

frothing of the blood, but no mechanical obstruction to the heart, without embolism in the lung and arterial circulation, and without any important or dangerous symptoms resulting therefrom. Dr. Senn could tell us how large quantities can be injected in the horse without producing bad effects.

I presume that this condition occurs in a large number of surgical cases. I remember performing tracheotomy on an adult female, in which the superficial veins were opened. I could hear the clicking of the air in the wound with each inspiration. I paid no attention to it, and no bad symptoms resulted. I presume that it can only be of serious import when some large sinus like the internal jugular vein has been largely opened, or if the longitudinal sinus or the axillary veins have been opened, and a large quantity of air has been sucked in by a sudden inspiration.

In speaking of the axilla, I may say that I have noticed aspiration of air into the axilla, which for the time being disturbed me, as it simulated somewhat this trouble. In the pumping efforts of the thorax, air may be drawn into the loose connective tissue of the axilla, producing a sound resembling that of the entrance of air into the vein.

Dr. Senn stated that this accident was more common in surgery before the days of anæsthesia than since. He ascribed that to the erect position favoring the entrance of air. It occurred to me that there might be another way of explaining the fact, if such it be, and that is, that the tension which was placed on the muscles of the patient under any painful operation and the gasping efforts at particularly painful periods, might impel the individual to take a sudden, short, sharp inspiration with all the muscles pulling in such a way as to open the veins and draw in several large bubbles of air. I should think some such explanation as that quite as possible as mere posture.

As I have said, it is in obstetrical practice that we meet with this accident in all its hideousness. In preparing for this discussion, I was reading over an interesting report on this subject by one of our medical examiners, Dr. F. W. Draper. Several cases of air-embolism following attempts to produce abortion have occurred in Boston. One was that of a woman several months advanced in pregnancy who went to an abortionist. She sat on a bed while he introduced an instrument into the uterus, and then passed a syringe

and began to pump in air, it is supposed. Air was forced in at any rate. In about fifteen minutes, she gave a low moan, gasped once or twice, and in half a minute was dead. At the autopsy, the right side of the heart was distended with air. The ascending cava was also filled with air. Air was found not only in these positions, but also in the capillaries of the lung and of the arterial system, so that when the sternum was cut through, air bubbled out from the arterioles of the anterior mediastinum. It was argued in that case, that, as the air had gone so far, it might have been possible that it had been introduced with a vaginal douche previous to coming to the abortionist's office. That was disposed of by the statement that the circulation makes its round in a few seconds, and air can be rapidly distributed in a short space of time. As Dr. Senn has mentioned, we have in such cases, conditions most favorable to the introduction of air into the circulation. A small amount of air gets into a uterus which is relaxing and contracting, there is a certain amount of hemorrhage with clots which may cork up the mouth of the uterus and prevent the escape of the air. Under such circumstances there would be a large number of holes, communicating with veins which have no valves, and affording a ready egress of air from the uterine cavity. In addition, we have the action of the thorax which tends to aspirate the air into the circulation. Therefore I say that this accident is more likely to occur in obstetrical than in surgical practice.

In regard to treatment, it seems to me that such a paper as Dr. Senn's is valuable in showing us the mechanical mode of development of such condition, and in showing us the conditions most favorable for its occurrence, and consequently telling us more how to prevent the accident than to help the patient after it has occurred. As one writer has said, the only function of the physician in such a case is to be present at the autopsy and confirm the diagnosis. That appears to be his sole duty.

Theoretically, aspiration of the heart seems to be indicated, but practically there probably never would be such a consensus of circumstances which would enable us to be on hand, to have the necessary instruments, and be quick enough in our diagnosis to enable us to use the instrument instantly. Such an accident is like a stroke of lightning. The patient is dead the moment the condition is recognized. The heart has been aspirated in other condi-

tions, and I suppose it could be in this. I do not see that there is much ground for comfort in this reflection, but in this, as in many other conditions, we should hope by a more perfect knowledge to avoid them, rather than to cure them.

Dr. Edward M. Moore, of Rochester, New York.

Mr. President, I should like to introduce to the Association, Dr. Gordon, of Portland, Maine, who will detail to us an extremely interesting case bearing on this subject.

Dr. Gordon, of Portland, Maine.

The allusion of Dr. Warren to Dr. Draper's case reminds me of an accident which occurred to me during the past winter, and inasmuch as we profit as much by our faults as by our virtues, it may be interesting to describe it.

In the hospital to which I am attached, the Maine General Hospital, I found, when I went on duty, a lady with pelvic abscess. Others had operated by aspiration, but had not found pus. A few days later, the development having gone on rapidly, I found no difficulty in drawing off a pint of ordinary pus. A week or ten days later, I did the same thing. In the mean time our house-surgeon had been changed, and a new one came on duty. I would say also that the aspirator used in the hospital was a bottle aspirator. Those who are familiar with it know that the pump will either condense or exhaust the air in the bottle, according as the tube is applied to one or the other nozzle. The new house-surgeon, who was not familiar with the instrument, attached the tube at the wrong end. I was attending to the needle, and entered it at exactly the same point as in the previous operations. When I was ready, I said, "turn on." He did so, and at the same instant the woman gave a scream, put her hand over her heart, and in half a minute was dead. The fact was that the bottle had been charged with air, the aspirating needle was introduced, I simply made the connection between the needle and the bottle, and the result followed.

Exactly what happened it is impossible to say. This cavity had been exhausted largely by the previous tappings, and the air may have entered through some of the vessels in the wall, or the needle may have been passed into a vein. The air certainly got in. This is one of those cases in which there is nothing to be done. I have

only this to say, that I shall never use such an instrument where it is possible for any one beside myself to have anything to do with it.

Dr. J. Ewing Mears, of Philadelphia, Pennsylvania.

Was an autopsy made?

Dr. Gordon. There was not. The husband objected decidedly to a post-mortem examination. I think that there can be no doubt that death was due to air-embolism. The symptoms were those which belong to that condition.

Dr. Charles B. Nancrede, of Philadelphia, Pennsylvania.

I rise with mingled feelings of admiration and regret to discuss this paper. The admiration is for the indefatigable energy of our Fellow, Dr. Senn, and the regret is that I have nothing especially new to offer on this very important, and I think rather neglected, subject. It is true, as he stated in his historical account, that it had been long known to physiologists that injection of air into a vein in sufficient quantity was fatal. The original idea was that a single bubble was fatal. The first recorded case is usually stated to be that of Beauchesne in 1818. It is said that the elder Larrey had frequently observed it on the battle-field from ball or sabre wounds, I cannot say which, but more likely the former. My authority for the statement is a systematic work on surgery, in which it is stated that Larrey said to Brodie, when looking at the picture of a man with a wound in the neck: "Ah! I know how that man died. I have frequently seen them die from aspiration of air into the veins on the battle-field." If my authority is reliable, Larrey antedates Beauchesne in his observations, although Beauchesne was the first to *record* this accident.

I think that it should always be borne in mind in performing experiments that animals are not human beings. I am free from any prejudice against experiments, for my graduating thesis was an experimental one, and I had the honor at the meeting in Cincinnati of laying before the Association the results of my experiments on inflammation in animals. As Dr. Senn mentions, death occurs in some animals with greater rapidity and ease than in others. In the horse it requires a large amount of air to produce a fatal effect. He explained this as being due to the strength of the right ventricle. There is an observation of Bérard which explains it better, especi-

ally if you bear in mind some other observations to which I shall refer. This author states that the pulmonary capillaries in the horse are much larger than in other animals. There are other experiments which show that if there is one thing pre-eminently necessary for free capillary circulation, it is a proper density of the blood. It was not mentioned in the portion of the paper read by Dr. Senn, that the injection of water is capable of producing symptoms identical to those produced by the injection of air, although, presumably in the former case, they pass away. The only explanation of this is a sudden alteration in the density of the blood preventing it from circulating in the capillaries of the lung. I think that the observation of Bérard in regard to the size of the capillaries of the horse, in connection with the fact that we can inject relatively large quantities of air into the veins of these animals, and that the air is found in the arterial circulation, showing that spumous blood will pass through the capillaries of the horse, is most important in connection with the therapeutics of this condition.

I cannot think from the history of the cases and from the experiments on animals, that death is a simple matter in air-embolism. It is due to a complication of causes, the chief being, I think, anæmia of the centres of respiration. Dr. Senn and other experimental observers have thrown large quantities of air into the heart, much larger than could be introduced in an operation before fatal symptoms would come on. Undoubtedly, in experimental cases, the heart is distended with air, but this does not obtain in the accidental entrance of air into the venous circulation during operation. The heart has been found over and over again not distended with air. It has been found to move with great force and strength even after respiration has ceased, so that death does not come from the heart, but from respiratory failure due to deficiency of arterial blood in the centres of respiration. Undoubtedly the inability of the right ventricle to act properly is due not only to the mass of spumous blood, which it is unable to pass on through the lung capillaries, but also to the fact that the changes in the density of the blood alters its physical conditions, and the valves cannot be forced back as they would be by dense blood. The result is that the blood is thrown backward and forward from auricle to ventricle. Under such circumstances the removal of spumous blood from the heart may be valuable.

Dr. Senn has called attention to the chief facts, which are ex-

planatory of the conditions favoring the entrance of air into the venous circulation, but there are others in operation which he does not note, such as the position of the neck or arm, stretching the axilla in the various operations required in that neighborhood. A slight amount of tension will keep a vein open, so that even the contractions of the platysma myoid muscle will do so to a certain extent. Traction on a tumor will do the same thing. There is another rare condition which it is possible to conceive of, viz., the division of a vein in the angle of a wound. As you raise the flaps you hold that vein open and effect a temporary canalization, and the evil results that follow this.

Dr. Senn made the remark that in cases where air was supposed to have been generated in the blood, the observation had been inaccurate, and that all the cases recorded were those in which free hemorrhage had taken place, and in consequence there were open vessels where air might have entered. I dissent from that. There are a certain number of cases where that explanation does not hold. I had a case of my own which I cannot explain in any other way, and there are a number of others on record, occurring chiefly in the course of septicæmia. I had a patient whose arm I had amputated at the shoulder-joint. Eight days later he sat up in bed apparently pretty well. He gave a sudden exclamation, and before the nurse could reach him he was dead. I could get no post-mortem examination. There was nothing that would kill him so quickly, except air-embolism. Deep pressure on the stump forced out bubbles of offensive gas. There are similar cases on record.

As Dr. Warren has said, the preventive treatment is more important than the curative. We cannot carry our whole armamentarium around with us, and we are not likely to have an aspirator convenient. I have always had a great dread of air-embolism, and there are certain things that I invariably aim to do in operating on the axilla or neck. Thus, I think that complete anæsthesia is most important. The sudden inspiration after a painful cut undoubtedly tends to draw air in. In passing, I would allude to the fact mentioned by Dr. Warren in reference to the sucking sound often heard during tracheotomy. I have heard this sound frequently, and have been in times past much alarmed at it, but I have come to the conclusion that it is due to the opening of the deep layer of the tracheal fascia. I have heard this sound when there were no open veins.

It is probably due to the same conditions which produce a similar sound when air enters the axillary tissues. Where anæsthesia is not used the arms and chest should be firmly bound down so that no sudden inspiratory movement can be taken. In removing a tumor from the neck, I usually tie its deep attachments, or hold them with clamp forceps on the cardiac side, before twisting off the tumor. If it is possible I never make a cut where there is any tension of the cervical or axillary tissues, unless there is a finger on the cardiac side of the vein.

Should air enter, what can be done? Experiment has shown that artificial respiration in those cases where a non-fatal quantity of air has been injected is of service. This should be done in the recumbent position. Air will not enter as readily in the recumbent position as in the erect position. The head should be dependent, the limbs raised, and artificial respiration carried on. Treves has recently suggested a method for preventing further entrance of air. It may not be easy to tie the vein, but a wet sponge can be at once squeezed, filling the wound with water. Then, as has been shown by experiments in animals and in the human subject where recovery has taken place, the symptoms are partly those of syncope. The arterial blood should be diverted from the extremities to the brain by compressing the axillary and femoral arteries. This expedient probably also effects another purpose. It increases the intra-cardiac pressure so that it is possible to have the valves closed and the blood forced through the pulmonary capillaries.

It is hardly necessary to say that the cardiac action should be kept up by stimulation. There is one suggestion that I should like to make, viz., in addition to the subcutaneous injection of ether and the application of ammonia to the nostrils and so on, we should use subcutaneous injections of atropia. If it is true that the injection of larger quantities of air are required to kill a horse, because of the larger size of the capillaries in that animal, the importance of dilating the human pulmonary capillaries to their fullest capacities is seen. This is a secondary result of a large dose of atropia. It also stimulates the respiratory centres. I have seen it do it. I reported a case last year in which the pulse and respiration had ceased for about three minutes. It was a case of brain abscess. I attributed the temporary recovery to the injection of atropia, life being prolonged for six days. It is indicated, and it can do no

harm. It is true that atropia does depress the action of the heart a little, but you get more than a compensating advantage by the enlargement of the pulmonary capillaries. Experience has shown, that if the patient can be kept alive for a short time he may recover. After recovery the patients are apt to be attacked with secondary pneumonia, which is to be treated on general principles.

Dr. Senn. I am under great obligations to the gentlemen who have so ably discussed my paper. Their remarks bear evidence of study and careful preparation. In view of the numerous well-authenticated cases of sudden death during surgical operations it requires no argument to prove that the subject of air-embolism should be as important to the surgeon as the obstetrician. The very fact that so many surgeons have witnessed this accident is a strong argument in favor of discussing this subject at this time and in this place with a view of arriving at some definite conclusions pointing towards preventing this occurrence, or in case a dangerous, or fatal amount of air has entered, what measures might be resorted to to modify or annul its deleterious effects. The introduction of a few bubbles of air does not produce death, because it neither interferes with the passage of blood through the pulmonary circulation nor with the perfect mechanism of the heart's action. When air is introduced slowly and in small quantities, not sufficient to arrest the heart's action, it is forced through the pulmonary capillaries into the left side of the heart and arterial circulation, where it is brought into contact with an extensive surface for absorption, and by its gradual removal sudden accumulation in dangerous localities is prevented. A clicking sound heard during an operation is no indication that air has entered a vein; such a sound may attend the ingress of air into any hollow space. The sound produced by the entrance of air into a vein resembles a sucking sound, which is best expressed by the German word "schluerfend." Muscular contractions always produce compression of a vein, and thus would rather tend to prevent, than to produce, entrance of air. When the necessary conditions are present to maintain the patency of a vein after incision I consider the elevated position the most important element in causing the entrance of air. The experiments on the superior longitudinal sinus reported in my paper sufficiently corroborate this statement. In regard to the treatment by direct aspiration of the right ventricle, which saved a good percentage of

the animals where a fatal dose of air had been administered, I must again emphasize the fact that over-distension of the right ventricle occurs as much from accumulation of venous blood from the obstruction in the pulmonary capillaries as from the presence of air, hence any procedure which can remove the cause of the over-distension in the shortest space of time must be relied upon in preventing paralysis by over-distension. The all-important indication presents itself to maintain the action of the heart until the obstruction in the pulmonary circulation has been removed. Death does not take place instantly. The heart often continues to contract feebly for some time, until by accumulation of blood in the right ventricle over-distension results in arrest of muscular contractions. As aspiration of the right ventricle in itself is not attended by any immediate or remote danger, I must insist that it should always be resorted to in cases of air-embolism when other measures are inadequate to prevent death by over-distension and paralysis of the heart. Removal of air from the right side of the heart by aspiration also constitutes the most direct measure in preventing death by asphyxia or acute cerebral anæmia, as it diminishes the number of air-emboli which otherwise would subsequently pass into and obstruct the pulmonary vessels. The experiments have also shown the usefulness of venesection in these cases, the beneficial effect being due to its direct effect in preventing engorgement and distension of the right side of the heart.

Air-embolism produces obstruction in the pulmonary vessels by bubbles of air becoming arrested in the vessels, which act, for the time being, like any other kind of emboli by mechanically interfering with the circulation. In the horse the contractions of the right side of the heart, as compared with other animals, are more powerful, and on this account over-distension is not as likely to take place, the air being forced rapidly through the pulmonary capillaries into the left side of the heart. Injections of air into the left side of the heart are less dangerous, because the powerful contractions of the left ventricle are more competent to force the air from the heart into the peripheral circulation and through the systemic capillaries. A simple alteration in the density of the blood is not attended by such grave symptoms indicative of obstruction in the pulmonary circulation as we observe it after air-embolism. Intravenous injection of a saline solution after sudden

and severe hemorrhage is never attended or followed by such a train of grave symptoms as we observe after the entrance of any considerable quantity of air into a vein. Auto-transfusion, as suggested by Dr. Nancrede, would, as he states, increase the intracardiac pressure, and on this account I should be afraid to increase the function of an already over-distended organ. The best possible method to remove the cerebral anæmia is to diminish the intracardiac pressure, as by doing so we maintain the action of the heart until the obstruction in the pulmonary vessels is removed, and the equilibrium between the arterial and venous circulation is restored. Theoretically the use of atropia would be indicated; practically, however, I should expect a more prompt effect from the inhalation of nitrite of amyl.

In closing my remarks I desire to thank the gentlemen for the interest they have manifested in my paper, and for the many valuable suggestions made.

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EXPERIMENTAL AND CLINICAL

STUDY OF AIR-EMBOLISM.

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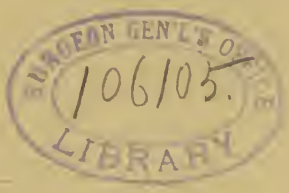

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